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PROCEEDINGS

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OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

VOLUME III

1905–1906

EDITED BY THE SECRETARY

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PREFACE.

In conformity with the custom inaugurated a year ago, this volume presents a brief biography of the second president of the society.

The constitution, as given on page 7 (177), includes the amendments that have been passed since the publication of volume II.

The numerals in parenthesis before the titles of the abstracts [page 15 (185)] indicate numerical positions in the entire series of communications presented before the society since its organization. These numerals are given on the assumption that some of the members desire to have bound together several (perhaps the first three) of these volumes. In such cases the numerical arrangement referred to, and that adopted for the index, will facilitate reference to the abstracts. The page numerals in parenthesis continue those of Volume II.

The numerals in the index at the end of the volume correspond with those in parenthesis before the titles of the abstracts. None of them duplicates any of the numerals in the index of volume I or of volume II.

September 1, 1906.

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Edmund B. Wilson

EDMUND BEECHER WILSON,

SECOND PRESIDENT (1905–'06) OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Edmund Beecher Wilson, professor of zoölogy at Columbia University, was born in Geneva, Illinois, October 19, 1856. After graduating from Yale College in 1878 with the degree of Ph.B., he spent three years at Johns Hopkins University, obtaining the Ph.D. degree in 1882. After devoting a year to study at Cambridge, Leipsic and Naples, he was lecturer in biology successively at Williams College and at the Massachusetts Institute of Technology, and was then called to Bryn Mawr College. Here he was for six years professor of biology, leaving this position for the chair which he has held for the past fifteen years at Columbia University.

Brought up in the country Professor Wilson early learned to know and to appreciate the things of nature, and developed a love for the common forms of animals and plants, which has never been lost in his later scientific work. In this he has not been satisfied with what may be termed the old-time problems of zoölogy; nor with petty details of technique; nor with the methods of the library naturalist. From the very outset of his scientific work his interests have centered in animals as living things. This has been the underlying factor in studies on development which he carried on for more than twenty years after obtaining his doctorate, and is still the fundamental principle underlying his researches on problems connected with the cell. His publications in the field of general zoölogy, as ordinarily understood, belong to the earlier period of his work, but they represent interests which, although subsidiary to those more absorbing ones which later claimed his attention, have never been given up. These later interests showed themselves first in his investigations on the history of the cleavage cells in the early stages of embryological development, some of which were published before he had taken his doctor's degree; and these papers gave evidence of the tendency, even at this early period, to turn to the cell for the ultimate analysis of vital phenomena.

These early studies, brought out at a time when cellular biology was in the period of anticipatory speculation, exerted wide influence in directing attention to the study of the laws of normal development from the standpoint of the cell, and they were followed later by equally stimulating studies in experimental embryology and experimental cytology which were carried out with special reference to the problems of mosaic development, prelocalization, and differentiation in the egg. During the last decade his interests have also turned to the mechanism of the cell and here again, his researches on the structure of protoplasm, on the history of centrosome, aster and karyokinetic figure, and on the chromosomes with special reference to the questions of heredity and sex, have had a wonderfully stimulating effect on biological research throughout the world. Many of these studies, too numerous to be listed here, were unified and worked up into harmonious relations with the modern aspects of the fundamental problems of biology in his book on *The Cell in Development and Inheritance*, which, through clearness of style and masterly critical analysis, has been one of the most widely read of modern technical scientific works, and is the one by which he is best known.

Professor Wilson's scientific attainments have been widely recognized in academic circles and by scientific societies. He is a member of the National Academy; a Fellow of all the general zoölogical societies of this country; of the Royal Microscopical Society of England, of the Accademia dei Lincei of Rome and of other societies. He received the LL.D. degree from Yale University and from the University of Chicago in 1901, and from Johns Hopkins University in 1902.

G. N. C.

CONSTITUTION AND BY-LAWS.

CONSTITUTION.

[As adopted February 25, 1903, and amended April 20, 1904, May 24, 1905 and April 18, 1906.][1]

Article I. Name.

The name of this organization shall be the Society for Experimental Biology and Medicine.

Article II. Object.

The object of this Society shall be the cultivation of the experimental method of investigation in the sciences of animal biology and medicine.

Article III. Membership.

Section 1. *Eligibility.* — Any person who has accomplished a meritorious original investigation in biology or medicine by the experimental method shall be eligible to membership.

Section 2. *Classification.* — The term "resident members" shall refer, in this constitution, to those members whose experimental work shall be done within the limits of "Greater New York"; "non-resident members," to those whose scientific work shall be done outside of "Greater New York."

Section. 3. *Obligations.* — A. Every member shall be expected to conduct an experimental investigation, and give public notice of it, at least once in two years.

B. *Resident* members shall be required either to attend, every two years, at least three meetings of the Society, or to present in person, at least once every two years, a report of their experimental researches.

C. Each *non-resident* member shall be required to present in person, at least once every two years, a communication containing

[1] The amendments adopted April 18, 1906 are indicated by heavy-faced letters, or by footnotes.

the results of an experimental investigation, or to send to the President within that time, such a communication for presentation at a regular meeting of the Society.

D.[1] Non-compliance with any of these requirements carries with it forfeiture of membership, unless an acceptable explanation is offered to the Council.

E. Any member of this Society who may consent to the use of his name in any way that would aid in increasing the sale of any patent medicine, proprietary food preparation, or any similar product known to be of doubtful value, shall forfeit his membership.

Section 4. *Nomination and Election.* — A. Each candidate for membership must be nominated by three members.

B. After their eligibility has been determined by the Council, nominees may be voted for at any meeting succeeding that at which their names were presented.

C. A three-fourths vote of the ballots cast shall elect.

Section 5. *Expulsion.* — Any member may be expelled by a three-fourths vote of the total membership.

Article IV. Meetings.

Section 1. *Time.* — The Society shall hold regular meetings at least once every two months during the academic year.

Section 2. *Annual Business.* — The first meeting held in the calendar year shall be the annual business meeting.

Section 3. *Program.* — The programs of the meetings shall consist of (A) brief presentations, in elementary form, of the essential points of experimental investigations, preferably demonstrations of actual experiments; and (B) of brief reports of important facts recently discovered in the sciences of biology and medicine or allied natural sciences.

Article V. Officials.

Section 1. *Officers.* — The officers shall be a President, a Vice President, a Secretary and a Treasurer.[2]

Section 2. *Council.* — The officers shall constitute the Council of the Society. Ex-Presidents of the Society shall be ex-officio permanent members of the Council.

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[1] Former section D was removed. See page 89 (259).

[2] The office of librarian was abolished. See page 89 (259).

SECTION 3. *Nomination and Election.* — A. Nominations of officers shall be made in the session immediately preceding the annual business meeting.

B. Election of officers shall be by ballot at the annual business meeting.

C. A plurality of the votes cast shall elect.

SECTION 4. *Term of Office.* — The term of office shall be one calendar year.

SECTION 5. *Duties.* — A. The duties of the officers shall be such as usually devolve on them individually, and also collectively, as an executive committee.

B. The Council shall promptly investigate and report its findings on the eligibility of candidates for membership.[1]

ARTICLE VI. DUES.

The annual dues shall be Two Dollars ($2.00), unless otherwise determined by the Council.

Non-payment of dues for three consecutive years carries with it forfeiture of membership.

ARTICLE VII. QUORUM.

Twenty members shall constitute a quorum for the transaction of business.

ARTICLE VIII. BY-LAWS.

By-laws may be adopted at any meeting by a majority vote.

ARTICLE IX. AMENDMENTS.

SECTION 1. Proposed amendments of the constitution must be endorsed by at least three members, at a regular meeting, and may be voted on at a succeeding meeting.

SECTION 2. It shall be the duty of the Secretary to give all members due notice of intended amendments.

SECTION 3. A two-thirds vote of the total membership, or a unanimous vote of the members present, shall be required for the adoption of an amendment.

[1] Former Section C was removed. See page 89 (259).

BY-LAWS.

[Adopted February 25, 1903, and amended May 24, 1905.]

I. *Meetings.* — A. The meetings shall be held on the third Wednesdays of October, December, February, April and May.

B. The meetings shall be opened at 8:15 p. m., and shall be closed at 10:30 p. m.

C. When possible the meetings shall take place in suitable laboratories.

II. *Time Allowed for Reports and Discussions.* — A. The time allowed for making individual communications, except demonstrations of experiments, shall be restricted to ten minutes.

B. Not more than five minutes shall be allowed to a member for the discussion of any communication.

III. *Order of Procedure to be followed at the regular meetings:*

A. Call to order.

B. Reading of minutes.

C. Report of council.

D. Scientific program.

E. Executive program.

- a. Reports of committees.
- b. Unfinished business.
- c. Election of members.
- d. Nominations for membership.
- e. New business.

F. Adjournment.

REGISTER OF NAMES AND ADDRESSES OF THE MEMBERS.

ABBOTT, ALEXANDER C.....University of Pennsylvania.
ABEL, JOHN J.....Johns Hopkins University.
ADAMI, J. GEORGE.....McGill University.
ADLER, ISAAC.....N. Y. Polyclinic Medical School.
ALSBERG, CARL L.....Harvard University.
ATKINSON, JAS. P.....Department of Health, New York City.
AUER, JOHN .....Rockefeller Institute for Medical Research.

BARDEEN, CHAS. R.....Wisconsin University.
BEEBE, S. P..... Cornell University Medical College.
BENEDICT, FRANCIS G.....Wesleyan University.
BRINCKERHOFF, WALTER R.....U. S. Public Health and Marine-Hospital Service, Honolulu.
BROOKS, HARLOW.....New York University.
BURTON-OPITZ, RUSSELL.....Columbia University.
BUXTON, B. H. .....Cornell University Medical College.

CALKINS, GARY N..... Columbia University.
CANNON, WALTER B.....Harvard University.
CARLSON, A. J.....Chicago University.
CASTLE, W. E..... ..... Harvard University.
CHITTENDEN, R. H.....Yale University.
CLOWES, G. H. A. .....Buffalo University.
CONKLIN, E. G.....University of Pennsylvania.
CRAMPTON, HENRY E.....Columbia University.
CRILE, GEO. W..... Western Reserve University.
CUNNINGHAM, RICHARD H.....Columbia University.
CUSHING, HARVEY W.....Johns Hopkins University.
CUSHNY, ARTHUR R.....University College, London.

DAVENPORT, CHAS. B..... Carnegie Institution's Station for Experimental Evolution, Cold Spring Harbor, Long Island, N. Y.
DAWSON, PERCY M.....Johns Hopkins University.
DONALDSON, H. H.....Wistar Institute of Anatomy, Philadelphia.
DUNHAM, EDWARD K.....New York University.

EDSALL, DAVID L.....University of Pennsylvania.
ELSER, W. J.....Cornell University Medical College.
EMERSON, HAVEN..... Columbia University.

Erlanger, Jos.....University of Wisconsin.
Ewing, Jas.....Cornell University Medical College.

Field, Cyrus W.....Department of Health, New York City.
Flexner, Simon..... Rockefeller Institute for Medical Research.
Flournoy, Thomas.....New York University.
Folin, Otto..... McLean Hospital, Waverly, Mass.
Foster, N. B.....Columbia University.

Gibson, Robert B.....Department of Health, New York City.
Gies, William J... .....Columbia University.

Harrison, R. G. .....Johns Hopkins University.
Hatcher, R. A.....Cornell University Medical College.
Hawk, Philip B.....University of Pennsylvania.
Hektoen, Ludvig.....Chicago University.
Henderson, Yandell.....Yale University.
Herter, Christian A.....Columbia University.
Hiss, Philip H.....Columbia University.
Howell, Wm. H.....Johns Hopkins University.
Huber, Carl G.....University of Michigan.
Hunt, Reid.....U. S. Public Health and Marine-Hospital Service, Hygienic Laboratory, Washington.

Jackson, Holmes C.....Albany Medical College.
Jennings, H. S.....Johns Hopkins University.
Jones, Walter..... Johns Hopkins University.
Jordan, E. O .....Chicago University.

Kastle, J. H.....U. S. Public Health and Marine-Hospital Service, Hygienic Laboratory, Washington.

Lee, Frederic S.....Columbia University.
Levene, P. A.....Rockefeller Institute for Medical Research.
Levin, Isaac.....Sydenham Hospital, New York City.
Lillie, Ralph S.....Johns Hopkins University.
Loeb, Jacques.....University of California.
Loeb, Leo.....University of Pennsylvania.
Loevenhart, A. S.....Johns Hopkins University.
Lombard, Warren P.....University of Michigan.
Lusk, Graham.....New York University.

Macallum, A. B.....University of Toronto.
MacCallum, W. G.....Johns Hopkins University.
MacDougal, D. T..... .....Carnegie Institution of Washington.
Macleod, J. J. R.....Western Reserve University.
Mandel, Arthur R.....New York University.
Mandel, John A.....New York University.

MATHEWS, ALBERT P.....Chicago University.
MELTZER, S. J.....Rockefeller Institute for Medical Research.
MENDEL, L. B.....Yale University.
MEYER, GUSTAVE M.....Columbia University.
MORGAN, T. H.....Columbia University.
MURLIN, J. R.....New York University.

NOGUCHI, HIDEYO.....Rockefeller Institute for Medical Research.
NORRIS, CHARLES.....Bellevue Hospital.
NOVY, FREDERICK G.....University of Michigan.

OERTEL, HORST.....Columbia University.
OPIE, E. L.....Rockefeller Institute for Medical Research.

PARK, WM. H.....New York University.
PARKER, G. H.....Harvard University.
PEARCE, RICHARD M.....Albany Medical College.
PFAFF, FRANZ.....Harvard University.
PORTER, W. T.....Harvard University.
PRATT, JOS. H.....Harvard University.

RICHARDS, ALFRED N.....Columbia University.

SALANT, WILLIAM.....Columbia University.
SCHWYZER, FRITZ.....St. Francis Hospital, New York.
SHAFFER, PHILIP.....Cornell University Medical College.
SHERMAN, H. C.....Columbia University.
SMITH, THEOBALD.....Harvard University.
SOLLMANN, TORALD.....Western Reserve University.
STEWART, G. N.....Chicago University.
STILES, PERCY G.....Massachusetts Institute of Technology.
STOOKEY, LYMAN B.....University of Southern California.
SYMMERS, DOUGLAS.....New York City Hospital.
SWEET, J. EDWIN.....Rockefeller Institute for Medical Research.

TAYLOR, ALONZO E.....University of California.
TERRY, B. T.....Rockefeller Institute for Medical Research.
TORREY, JOHN C.....Cornell Medical College.
TYZZER, E. E.....Harvard University.

UNDERHILL, FRANK P.....Yale University.

VAUGHAN, VICTOR C.....University of Michigan.

WADSWORTH, AUGUSTUS B.....Columbia University.
WALLACE, GEO. B.....New York University.
WARTHIN, ALDRED S.....University of Michigan.
WELCH, WM. H.....Johns Hopkins University.
WILLIAMS, HERBERT U.....University of Buffalo.

Wilson, Edmund B.....Columbia University.
Wolf, C. G. L.....Cornell University Medical College.
Wood, Francis C.....Columbia University.
Woodruff, L. L.....Williams College.
Woodworth, Robt. S.....Columbia University.

Yatsu, Naohidé.....Columbia University.
Yerkes, Robert M.....Harvard University.

Total number of members at the close of the academic year, 1905–'06..**119.**

OFFICERS.

Third year: February, 1905–February, 1906.

President.....Edmund B. Wilson.
Vice President.....Edward K. Dunham.
Librarian.....Graham Lusk.
Treasurer.....Gary N. Calkins.
Secretary.....William J. Gies.
Council—S. J. Meltzer, Edmund B. Wilson, Edward K. Dunham, Graham Lusk, Gary N. Calkins and William J. Gies.

Fourth year: February, 1906–February, 1907.

President.....Simon Flexner.
Vice President.....Edward K. Dunham.
Treasurer.....Gary N. Calkins.
Secretary.....William J. Gies.
Council—S. J. Meltzer, Edmund B. Wilson, Simon Flexner, Edward K. Dunham, Gary N. Calkins and William J. Gies.

SCIENTIFIC PROCEEDINGS.

Abstracts of the Communications.[1]

Thirteenth meeting.[2]

Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons. October 18, 1905. President Wilson in the chair.

1 (93).[3] **"A fatigue wheel": FREDERIC S. LEE.**

The author demonstrated a wheel designed for fatiguing mammals by means of voluntary muscular work.

2 (94). **"Mutation in the evening primrose, Onagra biennis (L.) Scop.,"** with demonstrations: **ELIZABETH BILLINGS** and **FREDERIC S. LEE.**

Culture experiments by the authors confirmed MacDougal's discovery of a narrow-leaved mutant of this species. From purely pollenated seed obtained by MacDougal and Britton from a wild plant growing at the New York Botanical Garden, 499 seedlings were obtained, of which 3 belonged to the narrow-leaved type. It is possible that a second mutant was found, but further observations are needed to confirm this. The species used by the authors is not *O. biennis* studied by de Vries.

3 (95). **"On the influence of thyroid feeding and of various foods and of small amounts of food upon poisoning by acetonitril": REID HUNT.** (Presented by **ALFRED N. RICHARDS.**)

One of the current theories of the functions of the thyroids is that these organs neutralize certain poisons occurring in the body; these poisons are purely hypothetical, and, so far as the author is aware, no one has yet reported experiments in which it has been shown that the thyroid can render a poison harmless. In the present experiments it was found that mice, to which thyroid had

[1] The authors of the communications have written the abstracts. The editor has made a few abbreviations and minor alterations in some of them.

[2] *Science*, 1905, xxii, p. 635; *American Medicine*, 1905, x, p. 911; *Medical News*, 1905, lxxxvii, p. 1143.

[3] See preface.

been fed for a few days, were markedly resistant to acetonitril; such mice recovered from the effects of ten to eleven times the ordinarily fatal dose of acetonitril. No such increased resistance to hydrocyanic acid or nitroprussiate of soda was caused by the thyroid feeding. Thyroidectin had an effect opposite to that of the thyroid, *i. e.*, it increased the susceptibility of mice to acetonitril, but this effect was not greater than that of dry normal blood and was less than that of peptone. Feeding with parathyroids had an effect opposite to that of thyroid, *i. e.*, it caused the mice to become more susceptible to acetonitril; the effect, however, was much less marked than that of the thyroid. Potassium iodid increased the resistance of mice to acetonitril, but the extent of this action was not at all comparable with that of thyroid.

In other experiments it was found that a protein diet (ham and cheese) caused an increased susceptibility of mice to acetonitril; a carbohydrate diet (rice and dextrose) increased the resistance to this poison. As a rule it required about four times as large a dose to kill the animals that were fed on a carbohydrate diet as it did to kill those fed on a protein diet. Animals kept on a very limited diet also showed a marked resistance to acetonitril; in most of such experiments it required about three times as much acetonitril to kill as was necessary to accomplish the same result on animals which had been kept on a normal diet.

The experiments are being continued.

4 (96). "**A case of spirochetal infection in man,**" with microscopical demonstrations: **Charles Norris.**

The author's object in presenting this case was to give the members of the society an opportunity of seeing spirochetas under the microscope. He did not discuss the clinical history of the case, which occurred in the service of Dr. Carlisle, of Bellevue Hospital.

In July, of this year (1905), the patient shipped as an assistant steward on the steamship *Denver*, of the Mallory line; he stayed five days in Galveston, sleeping on board, and returned on the same steamer to New York. Two days later he was taken with a chill, accompanied by fever, prostration, and pains in the bones. On admission he had a temperature of 102.4°. The fever continued for

two days. After four days of normal temperature, there was a rise of temperature to 105°, which was followed by a period of apyrexia for ten days, when he again had a relapse. At that time the examination of the blood by Dr. Heitlinger showed the presence of a few spirochetas. Ten days later there was another relapse and rise in temperature, associated with the presence of spirochetas in the blood. Inoculation of a monkey with blood containing the organisms gave rise to an infection, with the presence of spirochetas. The monkey has had three relapses thus far with rise of temperature, and the presence of spirochetas in the blood. Two additional monkeys have been infected with the blood of the first monkey.

The case reported is of interest from many points of view. It appears to be the first case of spirochetal infection reported in this country that was verified by microscopic examination of the blood. Another case, it is said, has been recently observed in one of the hospitals of this city.

The research work of the past few years, upon the tropic diseases of man and animals, has brought to light, especially in South Africa, the discovery of the etiologic agents of various hitherto little understood diseases.

Obermeier, in an epidemic of relapsing fever in 1868, in St. Petersburg, was the first to discover the presence of spirochetas in the blood of patients suffering from so-called relapsing fever. The observation was not published, however, until five years later. To Obermeier belongs the credit of having first demonstrated the so-called contagium vivum of infectious diseases in man. The association of spirochetas with another infectious disease was made by Sacharoff in 1890; he demonstrated the etiologic connection of *Spirochæte anserina* to the spirillum fever or septicemia of geese. In recent years, other spirochetas have been described in connection with disease processes. Thus, A. Theiler has described what he calls la spirillose du betail caused by a spirocheta which is found in the blood, where it produces an anemia, being present among cattle in a bad condition. Like the piroplasma bigeminum, it lives in the blood of immune cattle, as the disease has been inoculated with the blood of such cattle. The disease is conveyed through the agency of the blue tick, which is the intermediate

host. Like the piroplasma, the infective agent passes into the egg and is inoculated by the larvas. Theiler believes that this spirocheta is a parasitic protozoön.

Two English observers, Dr. Todd and the late Everett Dutton, have found that the tick fever, or at least some cases of tick fever, are associated with the presence of spirochetas in the circulating blood. They believe that tick fever is clinically identical with relapsing fever, and that its pathogenic agents are spirochetas, which they consider are probably identical with the spirochetas of relapsing fever, as described by Obermeier. They believe that a tick, *Ornithodorus moubata*, transmits the spirillum from animal to animal, since they have seen the disease conveyed to a monkey by a tick, and they have evidence that young ticks, after their first feeding, if bred from infected mothers, are able to transmit the disease. They have not been able to trace the spirilli in infected ticks further than the stomach and malpighian bodies. In the light of Marchoux and Salambeni's work, upon the transmission of the spirillum disease of fowls by ticks, Ross considers it probable that the disease in man is also inoculated by infected ticks.

It is unnecessary to enter at this time into the discussion of the protozoön nature of this interesting group of organisms, except to recall that Schaudinn believed there is little doubt that the spirochetas of relapsing fever and of the septicemia of geese will be shown to be trypanosomes, and hence unrelated to the bacteria. Novy and McNeal, it will be remembered, have shown, in a communication to this society,[1] that Schaudinn's interpretation of what he has seen is subject to grave doubt. The spirochetal forms of the trypanosomes depicted by Novy and McNeal, have not the slightest resemblance either to the organisms of this case or to Obermeier's or Sacharoff's spirilli, as shown by the photographs of the latter. The question as to the identity of the organism of this case, with that of the spirillum of Obermeier cannot be settled off-hand. On account of the great variety in the clinical symptoms of the reported cases of relapsing fever observed during the epidemics, it is perhaps unreasonable to draw any conclusions, either for or against the identity of the organism of this case with that of relapsing fever.

[1] *Proceedings* of this Society, 1904–'05, ii, p. 23.

To settle this question, morphology gives us little help. Although the organism of this case resembles the descriptions of the morphology as well as the photographs, of the spirillum of Obermeier, in practically all respects, it must be remembered that the spirillum of geese is strikingly similar to that of Obermeier, and yet, in the animal reactions, the anserina may be sharply differentiated from that of Obermeier, as it is not infective for monkeys.

The organism of this case, like Obermeier's, is infective for monkeys. The following differences have, however, been noted: The disease transmitted to the monkeys that were inoculated by the author seems to have been much milder than the experimental spirillum infection of those animals, as reported by various observers. Relapses in monkeys have rarely been noted; by one observer, in only one out of eight cases. Other observers seem never to have observed relapses. In the author's experience, each of three monkeys has had relapses, the first Rhesus having already had three.

Dr. Ewing has also called the author's attention to the fact that the spirochetas of this case, as seen in the blood of the inoculated monkeys, as well as in the human blood, is similar to *Spirochæte refringens.*

Such a case directs attention to the probability of mild spirochetal infections, more or less constantly occurring, in sailors or travelers coming from southern climates into the port of New York. The author also called attention to the possibility that infection may be communicated, from person to person, through the bites of ticks and bed-bugs, and through wounds.

5 (97). **"The chromosomes in relation to the determination of sex in insects": EDMUND B. WILSON.**

Material procured during the past summer (1905) demonstrates with great clearness that the sexes of Hemiptera show constant and characteristic differences in the chromosome groups, which are of such a nature as to leave no doubt that a definite connection of some kind between the chromosomes and the determination of sex exists in these animals. These differences are of two types. In one of these, the cells of the female possess one more chromosome than those of the male; in the other, both sexes possess the same number of chromosomes, but one of the chromosomes in the male

is much smaller than the corresponding one in the female (which is in agreement with the observations of Stevens on the beetle *Tenebrio*). These types may conveniently be designated as A and B, respectively. The essential facts have been determined in three genera of each type, namely (type A), *Protenor belfragei*, *Anasa tristis*, and *Alydus pilosulus*, and (type B), *Lygæus turcicus*, *Euschistus fissilis*, and *Cœnus delius*. The chromosome groups have been examined in the dividing oögonia and ovarian follicle cells of the female and in the dividing spermatogonia and investing cells of the testis in case of the male.

Type A includes those forms in which (as has been known since Henking's paper of 1890 on *Pyrrochoris*) the spermatozoa are of two classes, one of which contains one more chromosome (the so-called "accessory" or heterotropic chromosome) than the other. In this type the somatic number of chromosomes in the female is an even one, while the somatic number in the male is one less (hence an odd number), the actual numbers being in *Protenor* and *Alydus* ♀ 14, ♂ 13, and in *Anasa* ♀ 22, ♂ 21. A study of the chromosome groups in the two sexes brings out the following additional facts: In the cells of the female all the chromosomes may be arranged two by two to form pairs, each consisting of two chromosomes of equal size, as is most obvious in the beautiful chromosome groups of *Protenor*, where the size differences of the chromosomes are very marked. In the male all the chromosomes may be thus symmetrically paired with the exception of one which is without a mate. This chromosome is the "accessory" or heterotropic one; and it is a consequence of its unpaired character that it passes into only half the spermatozoa.

In type B all the spermatozoa contain the same number of chromosomes (half the somatic number in both sexes), but they are, nevertheless, of two classes, one of which contains a large and one a small "idiochromosome." Both sexes have the same somatic number of chromosomes (14 in the three examples mentioned above), but differ as follows: In the cells of the female (oögonia and follicle cells), all the chromosomes may, as in type A, be arranged two by two in equal pairs, and a small idiochromosome is not present. In the cells of the male, all but two may be thus equally paired. These two are the unequal idiochromo-

somes, and during the maturation process they are so distributed that the small one passes into one half of the spermatozoa, the large one into the other half.

These facts appear to admit of but one interpretation. Since all of the chromosomes in the female (oögonia) may be symmetrically paired, there can be no doubt that synapsis in this sex gives rise to the reduced number of symmetric bivalents, and that consequently all the eggs receive the same number of chromosomes. This number (11 in *Anasa*, 7 in *Protenor* or *Alydus*), is the same as that present in those spermatozoa that contain the "accessory" chromosome. It is evident that both forms of spermatozoa are functional, and that in type A, females are produced from eggs fertilized by spermatozoa that contain the "accessory" chromosome, while males are produced from eggs fertilized by spermatozoa that lack this chromosome (the reverse of the conjecture made by McClung). Thus if n be the somatic number in the female, $n/2$ is the number in all of the matured eggs, $n/2$ the number in half of the spermatozoa (namely, those that contain the "accessory") and $n/2-1$, the number in the other half. Accordingly:

In fertilization

$$\text{Egg}\,\frac{n}{2} + \text{spermatozoön}\,\frac{n}{2} = n \text{ (female).}$$

$$\text{Egg}\,\frac{n}{2} + \text{spermatozoön}\,\frac{n}{2} - 1 = n - 1 \text{ (male).}$$

The validity of this interpretation is completely established by the case of *Protenor*, where, as was first shown by Montgomery, the "accessory" is at every period unmistakably recognizable by its great size. The spermatogonial divisions invariably show but one such large chromosome, while an equal pair of exactly similar chromosomes appear in the oögonial divisions. One of these in the female must have been derived in fertilization from the egg-nucleus, the other (obviously the "accessory") from the sperm-nucleus. It is evident, therefore, that all the matured eggs must before fertilization contain a chromosome that is the maternal mate of the "accessory" of the male, and that females are produced from eggs fertilized by spermatozoa that contain a similar group (*i. e.*, those containing the "accessory"). The presence of but

one large chromosome (the "accessory") in the somatic nuclei of the male can only mean that males arise from eggs fertilized by spermatozoa that lack such a chromosome, and that the single "accessory" of the male is derived in fertilization from the egg-nucleus.

In type B all the eggs must contain a chromosome corresponding to the large idiochromosome of the male. Upon fertilization by a spermatozoön containing the large idiochromosome a female is produced, while fertilization by a spermatozoön containing the small one produces a male.

The two types distinguished above may readily be reduced to one; for if the small idiochromosome of type B be supposed to disappear, the phenomena become identical with those in type A. There can be little doubt that such has been the actual origin of the latter type, and that the "accessory" chromosome was originally a large idiochromosome, its smaller mate having vanished. The unpaired character of the "accessory" chromosome thus finds a complete explanation, and its behavior loses its apparently anomalous character.

The foregoing facts irresistibly lead to the conclusion that a causal connection of some kind exists between the chromosomes and the determination of sex; and at first thought they naturally suggest the conclusion that the diochromosomes and heterotropic chromosomes are actually sex determinants, as was conjectured by McClung in case of the "accessory" chromosome. Analysis will show, however, that great, if not insuperable, difficulties are encountered by any form of the assumption that these chromosomes are specifically male or female sex determinants. It is more probable, for reasons that will be set forth hereafter, that the difference between eggs and spermatozoa is primarily due to differences of degree or intensity, rather than of kind, in the activity of the chromosome groups in the two sexes; and we may here find a clue to a general theory of sex determination that will accord with the facts observed in Hemiptera. A significant fact that bears on this question is that in both types the two sexes differ in respect to the behavior of the idiochromosomes or "accessory" chromosomes during the synaptic and growth periods, these chromosomes assuming in the male the form of condensed chromosome

nucleoli, while in the female they remain, like the other chromosomes, in a diffused condition. This indicates that during these periods these chromosomes play a more active part in the metabolism of the cell in the female than in the male. The primary factor in the differentiation of the germ cells may, therefore, be a matter of metabolism, perhaps one of growth.

6 (98). **"Experimental hepatic cirrhosis in dogs from repeated inhalations of chloroform": C. A. HERTER** and **WM. R. WILLIAMS.**

The difficulty of inducing pronounced interstitial hepatitis in dogs by means of poisons makes it of interest to report the well-defined results obtained as a consequence of repeated inhalations of chloroform vapor. Experiments of this character were made upon three dogs. In one experiment the animal received chloroform three times a week on eighteen occasions, each inhalation having been continued for an hour. For six subsequent inhalations the duration of the narcosis was one and a half hour. The duration of the entire experiment was about eight weeks. The liver everywhere was found to be the seat of an abundant, richly cellular, connective tissue growth between and into the lobules. The bile ducts were proliferated, and the liver cells showed much fatty and hyaline degeneration.

In two other dogs similar experiments were carried out with the exception that in each of these instances a highly satisfactory control was secured by first removing a small portion of normal liver for subsequent comparison with the damaged liver. In one of these dogs the inhalations were given eighteen times in about six weeks. The animal lived somewhat longer than five months and showed a well-marked though not extreme cirrhosis. The third dog was narcotized forty-nine times and lived about eight months. The changes in this instance were perfectly distinct, but less advanced than in either of the other animals mentioned.

The liver tissue from the first dog was subjected to an analysis which showed a distinct fall in the normal percentage of the arginin constituent of the protein molecule. Similar analyses show that the arginin yield from protein may fall rapidly after even very short exposure to toxic influences and these results, indicating early

damage to living protoplasm, give much force to the contention that the connective tissue overgrowth in these cases of hepatic cirrhosis is secondary to changes in the chemical constitution of the liver cell. A further feature of interest is the fact that in two of the dogs the liver cells contained little fat at the time of autopsy. Finally, it may be mentioned that although a considerable loss in weight was observed in the dogs during the period of repeated narcotization, this loss was subsequently recovered in spite of the persistent cirrhotic changes.

These observations open the question whether the fatty and parenchymatous degenerations of the liver, which in some cases follow narcosis by chloroform in the human subject, may not occasionally pass on to interstitial cirrhosis — a single narcosis in man being sufficient to induce the primary damage to the protoplasm of the liver cell.

7 (99). **"Color sense in different races of mankind": R. S. Woodworth.**

The evolution of the color sense is very imperfectly understood. Scarcely any direct evidence is at hand regarding the color sense of animals, though some indirect evidence that various classes distinguish colors is afforded by the facts of protective and attractive coloration.[1] We do know from human experience, that there exists a form of color vision (red-green blindness) which is less complete than the usual human type, and as it appears not to be pathologic, it may be a reversion. In the absence of subhuman data, it is of some value to ascertain whether those races of mankind which seem to represent the more primitive stages in human development are especially subject to color-blindness. The results of various authors go to show that other races are perhaps even less subject to it than the white race. Some previously untested races were examined by the author in association with Mr. Frank G. Bruner, under the Anthropological Department of the St. Louis Exposition. Of 252 adult male Filipinos (including Christians and Moros), 14 were red-green blind, or 5.6 per cent.; of 75 males of the "wild tribes" of the Philippines (Igorots, Tinguianes and Bagobos), 2 were red-green blind, or 2.7 per cent.; of

[1] See Grant Allen: The Color Sense, Its Origin and Development, 1879. W. A. Nagel: Der Farbensinn der Tiere; Wiesbaden, J. F. Bergmann, 1901.

13 male Negritos, none was color blind. Special interest attaches to the Negritos, as they probably represent a more primitive type of man than has previously been tested in this way; and though the individuals examined were too few in number to enable the author to establish the percentage of color-blindness among them, the absence of color-blindness from the 13 males tested (as well as from the women) shows certainly that color-blindness is not universal among them, and very likely no more prevalent than among more developed races. On the whole a negative conclusion is warranted as to the suggestion that the color sense has developed, within human history, from anything approaching red-green blindness.

Quite a different hypothesis has been advanced by certain anthropologists from a study of the color names of primitive languages. While all languages have names for red, and most of them also for yellow, comparatively few have definite names for green, blue, or violet. Even in European languages, the names of these latter colors seem to be a rather recent acquisition. The suggestion is that color vision was first developed for the red end of the spectrum, the rest remaining colorless at first, and only gradually taking on the appearance of green and blue, and that this development has occurred during human history. In testing the natives of Torres Straits, who have no name of their own for blue, Rivers obtained a certain amount of evidence in favor of this view, in that these people were somewhat less sensitive to faint tints of blue than Europeans, though rather more sensitive to red. As the Filipinos also have no native words for green, blue and violet, the authors tested them as to their power of discriminating these colors. The test employed called for the matching of dark shades of several colors with pale tints of the same. Colored papers were used; the tints were spread out in spectral order, and each dark shade was to be matched with the tint with which it agreed in color. The authors found that the Filipinos, and indeed all other races examined, were inferior to whites in this test; but it was impossible to detect any special deficiency for the greens, blues and violets. These colors were relatively as well matched as the reds, and better than the yellows. Nor was there any tendency, except among the Igorots, to confuse blue, green or violet with neutral gray.

The Negritos did better than many more advanced races. The results obtained by the author are thus opposed to the view that the color sense has developed within human history from a more primitive type, in which only the red end of the spectrum appeared as colored.

8 (100). **"The practical concentration of diphtheria antitoxin": R. B. Gibson.**

The methods which have been proposed for the purification or concentration of antitoxins are, for the most part, peculiar and tedious ways by which the whole or a portion of the globulins are separated from serum or milk. Evaporation and freezing have been tried, but the general use of such methods has not been continued. Pick states that by the isolation of his soluble or high ammonium sulfate fraction, it is possible to concentrate the protective properties several times. Though superficially the most applicable, Pick's method is open to certain objections. Considerable quantities of antitoxin may be carried down with the nonprotective fraction on one-third saturation of the serum with ammonium sulfate. Such a concentration is also not practicable.

An artificial concentration can best be effected, for the present at least, by preliminary isolation of the antitoxin globulins; on this procedure is based the plan of the following method which has proved fairly successful.

The serum is precipitated with an equal volume of saturated ammonium sulfate solution and, after reprecipitation, is extracted with a solution of saturated commercial sodium chlorid. The antitoxic globulin is easily dissolved in the chlorid solution. The nonsoluble globulin settles to the bottom on standing. After filtering, the NaCl solution of the antitoxic globulin is precipitated by the addition of a half volume of saturated ammonium sulfate solution, or better still, with acetic acid in the usual way. The filtered precipitate is pressed as dry as possible with paper and dialyzed in parchment a few hours. Its solution is then neutralized and dialyzed again in running water. After two or three days' dialysis of the neutralized solution of the protein precipitate, sterilization is accomplished by double filtration through a Berkefeld filter. Before filtration, sufficient sodium chlorid is added to make its proportion equal to

0.5 per cent., and a preservative is used. The strength of the filtered product is ascertained. It is tested bacteriologically, injected into animals and finally actually administered in the Department of Health hospitals before distributing.

By this method almost all the ammonium sulfate is removed before dialysis, and the additional acid precipitation gives a purer product. Dialysis is quicker under these circumstances than when the sulfate alone is employed to effect precipitation. The antitoxin is practically all recovered, and a concentration of several times the original potency is easily and constantly obtained. The sodium chlorid separation is sharp, the two groups of proteins showing essentially different physical characters as precipitates. The final product is somewhat viscous, faintly opalescent and colorless or slightly tinged with hemoglobin. Dried at low temperatures, a beautifully transparent and entirely soluble scale antitoxin is obtained. Large quantities of serum can easily be worked over in this way at comparatively small expense.

Tests show that the artificially concentrated antitoxin, kept in small vials in an icebox, preserves its potency as well as or even better than the ordinary antitoxic serum. Therapeutically, the comparative results obtained are identical. Local irritation, rashes, etc., seem to be less frequent and severe when the refined antitoxin is administered.

9 (101). **"On the effect of magnesium salts upon the excitability and conductivity of nerves": S. J. MELTZER** and **JOHN AUER.**

In their communication to this society on the anesthetic effect of magnesium salts after subcutaneous injections,[1] the authors stated that they made several series of experiments on the physiological and pharmacological effects of these salts and that all their experiments had demonstrated a common result, namely, that magnesium salts produce a profound effect upon the nervous system and that this effect is invariably of an inhibitory character.

In their recent experiments the authors applied solutions of magnesium salts to the sciatic, pneumogastric, depressor, and sympathetic nerves of rabbits. Numerous applications of the magne-

[1] *Proceedings* of this Society, 1904–'05, ii, p. 81.

sium salts to the various nerves failed to produce, in any instance, a phenomenon which could be interpreted as an excitation, but in all cases there was produced, sooner or later, a profound inhibitory effect upon the conductivity of the nerve under experimentation. After application to the sciatic nerve, the conduction of motor and sensory impulses was manifestly inhibited; a strong stimulus applied below the block caused strong contractions of the muscles of the thigh, but no pain; when applied above, pain but no contraction was caused. In experiments on the depressor, stimulation on the distal side of the block failed to produce a fall of blood-pressure. Applications to the sympathetic blocked the conductivity, so that strong electric stimulations applied to the section of the nerve exposed to the influence of the solution, or distal to that section, failed to cause a constriction of the ear vessels or a dilation of the pupil.

Instructive results were obtained in the experiments upon the vagi. As is well known stimulation of the central cut end of the vagus produces an unmistakable effect upon respiration, while stimulation of the peripheral cut end causes a standstill of the heart, and a contraction of the esophagus and the cardia. When a section of about 2 cm. to 3 cm. of an intact vagus was exposed for some time to the influence of a solution of a magnesium salt, stimulation above the block affected the respiration but not the heart or the esophagus, and stimulation below affected the function of the last named organs but not that of respiration.

Applications to the vagus nerves enabled the authors to study the blocking of *normal* impulses. When applied to one nerve, after the other had been cut, or when applied to both intact nerves, the respirations slowed up perceptibly after a while, as happens after cutting both vagi; besides, after spontaneous or induced acts of deglutition, no contractions of the esophagus or cardia followed.

These effects were obtained with hypertonic as well as with isotonic, and even with strongly hypotonic solutions. The weaker the solution the longer it took to establish a complete block; 10 to 30 minutes was about the average time. After a block was established, conductivity could be completely restored by thorough irrigation of the nerve with Ringer solution. When hypotonic solutions of magnesium salts were used, conductivity was often

restored spontaneously without washing with Ringer solution, if the application of the magnesium solution was not renewed. After the application for two or three hours of strongly hypertonic solutions, the conductivity did not return usually for 24 hours or longer, sometimes not even after washing with Ringer solution, as was observed in experiments on the sciatic and on the superior cervical ganglion. Thus far the experiences of the authors in this connection indicate, however, that conductivity is finally restored in all cases.

Fourteenth meeting.[1]

Rockefeller Institute for Medical Research. December 20, 1905. President Wilson in the chair.

10 (102). **"The action of eosin upon tetanus-toxin and tetanus": SIMON FLEXNER** and **HIDEYO NOGUCHI.**

Eosin and certain other anilin dyes have the power of destroying in vitro the hemolytic property of tetanus-toxin.

Eosin, when used in sufficient quantity, destroys tetanospasmin in vitro.

Simultaneous injection of tetanus-toxin and eosin into rats delays or prevents the appearance of the symptoms of tetanus. When the symptoms appear they progress more slowly than in control animals.

Spores of tetanus-bacilli when introduced on threads into rats, and followed immediately by an injection of eosin into the same locality, do not produce tetanus. The treatment of animals with eosin, after the first appearance of the tetanic symptoms following spore-infection, may prevent the further development of the symptoms of tetanus. Eosin injections into the same locality as the spore inoculations are the most effective, but injections into other parts of the body delay or modify the tetanic process.

Rats are more resistant to tetanus poison than guinea-pigs, and hence are more easily protected by eosin from tetanus; but in guinea-pigs the fatal issue can be delayed by eosin.

[1] *Science*, 1906, xxiii, p. 109; *American Medicine*, 1906, xi, p. 105.

11 (103). "**The action of eosin and erythrosin upon snake venom**," with demonstrations: **Hideyo Noguchi**. (Communicated by **Simon Flexner**.)

The hemolytic principles of venom react differently to eosin depending upon their native labilities. The hemolysin of Crotalus venom suffers most; that of Daboia next, while that of Cobra is the most resistant.

The toxicity of different venoms is more or less diminished by eosin in the light. Cobra is least affected; Crotalus and Daboia venoms are most affected. Crotalus venom loses its toxicity chiefly by destruction of hemorrhagin, and Daboia by destruction of coagulin.

Neurotoxin is little or not at all affected by eosin or erythrosin.

There is a parallel between the susceptibility of the toxic principles of snake venom to fluorescent anilins and to other injurious influences. Hemorrhagin and coagulin are less stable at high temperatures than neurotoxin, and more easily destroyed by acids than neurotoxin and hematoxin.

12 (104). "**On the decomposition of purin bodies by animal tissues**": **P. A. Levene** and **W. A. Beatty**.

The authors aimed in this work to study the products of decomposition of purin bodies in the tissues. Jones, Schittenhelm and Levene have observed that aminopurins are transformed into oxypurins. It is well known that purin bodies undergo complete destruction in the course of tissue autolysis.

The authors have studied the conditions most favorable for the process of purin decomposition by animal tissues, and have endeavored to ascertain the general nature of the substances formed during the process. It was found that the presence of 0.5 per cent. of sodium carbonate in mixtures of spleen pulp facilitated the decomposition of purin bodies to such an extent that even uric acid was broken up by that tissue. It was also noticed that the decomposition products were nonbasic in nature, for they were not precipitated by phosphotungstic acid. On the decomposition of uric acid by tissue extracts, a formation of ammonia could not be detected.

13 (105). "**On the biological relationship of nucleoprotein, amyloid and mucoid**": **P. A. LEVENE** and **JOHN A. MANDEL.**

The authors endeavored to ascertain the nature of the carbohydrate groups in the protein molecule. It was found that by heating nucleoprotein on a water bath with a 5 per cent. solution of sulfuric acid, a product could be obtained that had the properties of a polysaccharid or of a glucosid, and which contained in its molecule a small proportion of sulfuric acid (S = 0.5 per cent.). On treating nucleoproteins with alkali, substances were obtained containing a much greater proportion of sulfuric acid (S = 3.5 per cent.; N = 8.8 per cent.). The substances thus obtained were found to possess the properties of glucothionic acids containing small quantities of nucleic acid.

Glucothionic acid has hitherto been recognized as a constituent of mucoid and amyloid. The results of this investigation place the three groups of substances in genetic relationship.

14 (106). "**On the imperfection of Mendelian dominance in poultry hybrids,**" with demonstrations of photographs and plumage-charts: **C. B. DAVENPORT.**

According to the Mendelian formula one of the pair of characters that are opposed in hybridization dominates over the other, occluding it; the dominated, or recessive, character reappears in its pristine purity when the hybrids are interbred.

A careful examination of the facts shows that in poultry hybrids the dominant character is frequently modified by the presence of the recessive and in the direction of the latter. For example, white plumage color may dominate over black, but the white hybrid shows some black feathers; white dominates over buff plumage, but the hybrids have a buff cast. Pea comb is dominant over single, but the middle lobe of the hybrids is unusually high. Narrow nostril is dominant over the high nostril of the Polish fowl, but the hybrid nostril is exceptionally wide. When the hybrids are interbred the recessive character reappears in about one-fourth of the hybrids, but often so modified as to be scarcely recognizable. The gorgeous bright red and golden but recessive plumage of the Japanese long-tailed fowl reappears in the second hybrid generation as a dull brick red, much mottled with black. The fact of the

mutual contamination of characters in hybrids justifies the warnings given by breeders as to loss of characters in hybridization, and the care that they exercise to maintain pure races.

15 (107). **"The mechanism of conduction and coördination in the heart, with special reference to the heart of Limulus": A. J. Carlson.** (Presented by **Russell Burton-Opitz.**)

I. *The Rate of Conduction.* — It is advocated, chiefly by Engelmann, that the rate of conduction of an impulse in the heart is too low (20 cm. to 30 cm. per sec. in the frog; 2 m. to 4 m. per sec. in the dog) to take place in the nervous tissue. The slow conduction in the heart is thus construed as an argument in favor of the myogenic theory. This is based on the erroneous assumption that all nervous paths in the same animal conduct with the same, or practically the same, rapidity. The author has shown that this is not the case even for the motor nerves to the striated muscles. On the contrary the rate of conduction in the nerve stands in direct relation to the rapidity of contraction of the muscle supplied by the nerve.[1] On this principle one would expect the *rate of conduction* in the intrinsic nervous plexuses of the alimentary tract and of the heart of a vertebrate to be as much slower than that in the motor nerves to the skeletal muscles, as the *contraction* of heart-muscle and muscle of the digestive tract is slower than that of skeletal muscle. The rate of conduction in the intrinsic nerves of the vertebrate heart has not yet been determined. In the heart of Limulus, this can be done by the ordinary graphic method. The author has shown that in the heart of Limulus the rhythm is neurogenic, not myogenic, and that the conduction and coördination take place in the nervous and not in the muscular tissue.[2] The proofs of these conclusions are demonstrative. The author has lately measured the rate of conduction in the intrinsic heart nerves of this animal and has found it to be 40 cm. per second. The rate in the motor nerves to the limbs as found by the author is 325 cm. to 350 cm. per second. That is to say, *the rate of conduction in the nervous plexus in the heart is from eight to ten times slower than in the peripheral motor nerves.*

[1] Carlson: *American Journal of Physiology*, 1904, x, p. 401.

[2] Carlson: *American Journal of Physiology*, 1904–'05, xii, p. 67; also, p. 471.

II. *Conduction in the Heart in the State of Water-Rigor.* — The experiments of Fredericq, Waller and Reid, Bayliss and Starling, Schlüter, Engelmann, Hofmann, and Bethe have shown that the heart walls may conduct without contracting or being able to contract. This can be interpreted in two ways, viz.: (1) The conduction takes place in the nervous tissue, or (2) the conduction takes place in the muscular tissue, but the processes of conduction and contraction are so independent of one another that the muscle may conduct without contracting. The latter is the explanation usually adopted, based on the experiments of Biedermann and Engelmann on conduction in muscle in the state of water-rigor. Engelmann worked on the frog's heart. In the heart of Limulus the above two possible explanations may be put to experimental test.

The author transected the heart-muscle in the region of the second and the fourth heart-segments and dissected away a portion of the muscle about 0.5 cm. in length, leaving the three portions of the heart connected alone by the nerve-plexus (the median nerve-cord and the lateral nerves). The anterior and the middle portions of the heart continued in rhythm by virtue of the impulses from the ganglion of the posterior portion, these impulses reaching the two anterior portions by means of the intact nerve-plexus. When this nervous plexus is severed in the fourth segment, the region of the heart anterior to the sections ceases to beat. Hence, the anterior portion of the heart thus prepared beats in response to impulses that reach it through the nerve-plexus on the middle portion. Now, when this middle portion of the heart is placed in water, the muscle of this region absorbs water and ceases to beat or respond to artificial stimulation, while the anterior portion still beats in synchrony with the posterior portion of the heart. The nerves will also lose their conductivity if left in the water long enough. On replacing the water by plasma or sea-water the nerves are quickly restored. The muscle is restored very slowly and sometimes not at all. The nerve-plexus in the Limulus heart is composed of nonmedullated nerves, just as is the intramuscular nerve-plexus in the heart of a vertebrate. Now, since the behavior of the Limulus heart and the heart of a vertebrate in the state of water-rigor is the same, and, further, as

the anatomic conditions (nerve-plexus and muscle-cells) are similar in both, it seems probable that the tissue concerned with conduction in water-rigor is also the same in both. In the Limulus heart it has been demonstrated to be the nerve-plexus and not the muscle. In the vertebrate heart it has not been demonstrated to be the muscle. The recent experiments of Humblet, Hering, and Erlanger, of transecting on compressing the auriculoventricular muscle-bundle in the septum of the mammalian heart, decide nothing relative to the myogenic or neurogenic nature of conduction and coördination, because it has been shown by Tawara that this muscle-bundle is surrounded and accompanied by a nerve-plexus similar to that in the auricles and the ventricles themselves.

16 (108). **"Further observations on the effects of alcohol on the secretion of bile": William Salant.**

In a previous communication[1] on the effect of alcohol on the secretion of bile, it was stated that diminution in the rate of secretion of bile was observed after intravenous injection of alcohol. No definite conclusions could be reached at that time, however, as to whether the diminished secretion was due to alcohol, for a steady decline in the flow of bile was very often noticed during the periods before the administration of alcohol. Recent observations in a series of similar experiments on dogs, in which the rate of secretion remained unchanged for several periods or differed slightly, showed some diminution of the flow of bile after intravenous injection of alcohol. There was also a decrease in both the organic and inorganic constituents of the bile after intravenous injection of alcohol, but the relative amounts of solids were only slightly affected. The diminished excretion of solids, however, cannot be attributed to alcohol, for a wide range of variation prevails in the organic and inorganic constituents of the bile of untreated animals.

The effects are entirely different when alcohol is introduced into the gastrointestinal canal. The methods employed in this relation were identical with those of the previous experiments. Anesthesia was induced by ether without the aid of morphin. In every case the neck of the gallbladder was securely ligated to prevent flow of bile from that direction. A cannula was then intro-

[1] *Proceedings* of this Society, 1904, i, p. 43.

duced into the common bile duct and the rate of secretion studied by comparing the quantities collected for periods of 15 minutes each. In one experiment in which secretion proved to be very scanty, the bile was collected for an hour and the quantity obtained during that period was compared with the amounts collected for equal lengths of time after injection of alcohol. Various strengths of alcohol were used: 25 per cent., 30 per cent., 50 per cent., 60 per cent., in quantities ranging from 1 c.c. to 5 c.c. per kilo, administered 1 to 2¼ hours after the introduction of the cannula into the common duct.

With the exception of experiment XI in the accompanying table (I), the volume secreted immediately after the injection of alcohol into the stomach or into the intestines showed a marked increase as compared with the period immediately preceding the injection of alcohol. In 11 of the 12 experiments performed on different dogs, the percentage of increase, as shown in the accompanying table (I), ranged from 50 per cent. to 365 per cent. In a large proportion of the experiments, in which the dogs were apparently so exhausted that the secretion of bile reached a minimum, the introduction of alcohol into the stomach or intestine caused a striking improvement. In some experiments alcohol was injected both intravenously and into the intestines. The volume of bile secreted after the intravenous injection indicated a diminished rate of secretion, while in the same animal after the administration of alcohol into the intestines the secreted volume of bile increased 140 per cent. in one experiment and 80 per cent. in another. The solid constituents were likewise markedly increased. In one experiment there was an increase of 130 per cent. in the total solids, 132 per cent. increase of organic matter, and 115 per cent. increase in the ash, the increase in volume in the same experiment being 140 per cent. In another experiment the total solids increased about 100 per cent., organic matter 108 per cent., and ash 60 per cent., the gain in volume being 125 per cent. Of two experiments, the increase in the volume secreted as well as in the amounts of solid constituents was 80 per cent. in one; in the other, the figures showing percentage increases in the secreted volume, total solids, organic matter, and ash were 160, 185, 195, 112, respectively, indicating that, at least in certain cases, some of the

solid constituents may be increased in amount, both absolutely and even relatively, after administration of alcohol. In this instance alcohol was introduced into the stomach. The excretion of inorganic constituents, while showing a well marked increase after the injection of alcohol into the gastrointestinal canal, did not keep pace with the gain in proportion of organic matter.

Further study is in progress.

Table I. — Effects of Alcohol, Injected into the Gastrointestinal Canal, on the Elimination of Bile (Collected in 15-minute Periods).

No.	Volume before injection of alcohol into the gastrointestinal canal.	After injection of alcohol into the gastrointestinal canal.	
		Volume.	Percentage increase.
	c.c.	c.c.	
I	0.5	1.2	140
II	0.5	0.9	80
III	0.5 (1 hr.)	1.0 (1 hr.)	100
IV	0.15	0.7	365
V	0.7	1.8	160
VI	0.4	0.9	125
VII	0.1	0.4	300
VIII	0.2	0.6	100
IX	0.3	0.5	66
X	0.3	0.45	50
XI	1.3	1.3	—
XII	0.25	0.5	100

Table II. — Effects of Alcohol, injected into the Gastrointestinal Canal, on the Elimination of Solids in the Bile (Collected in 15-minute Periods).

No.	Before injection.			After injection.			Percent. increase after injection.			
	Vol.	Total solids.	Ash.	Vol.	Total solids.	Ash.	Vol.	Total solids.	Organic matter.[1]	Inorganic matter.
	c.c.	mg.	mg.	c.c.	mg.	mg.				
I	0.5	34.2	4.6	1.2	78.8	9.9	140	130	132	115
II	0.5	48.6	1.2[2]	0.9	87.6	11.8	80	80	—	—
III	0.7	74.0	9.3	1.8	211.1	19.7	160	185	195	112
IV	0.4	42.3	6.4	0.9	85.1	10.2	125	100	108	60

17 (109). **"Some effects on rabbits of intravenous injections of nicotin,"** with demonstrations: **I. Adler** and **O. Hensel.**

A solution of 1 in 200 of the chemically pure nicotin furnished by Merck was used. Of this solution, ⅓ of a c.c., equal to 1½

[1] Calculated by difference from the total solids. The weights of organic matter are purposely omitted from the first two sections of the table.

[2] Probably some analytic error accounts for this anomalous result.

mg. of nicotin, was injected daily into the ear-vein of the rabbits. About ten seconds after such an injection the animal is seized with a typical convulsion lasting from three to five minutes, after which it is apparently entirely well until the next injection, when the same thing recurs. This may be repeated with great regularity and without any exception every day and no tolerance to the poison seems to develop. In two animals it was attempted gradually to increase the daily dose to ½ c.c. This, however, proved too dangerous and was abandoned. All animals thereafter received the same daily dose of ⅓ c.c., which was never increased or diminished. A number of animals died before they had received a sufficiently large number of injections to cause any definite lesion. Death ensued in some instances from some cause not at all referable to the nicotin poisoning, but in others from numerous small infarctions in the lungs, possibly caused by the intravenous injections. Cerebral hemorrhages, which are found so often in rabbits treated with adrenalin injections, were never found in these animals.

In animals which outlived a certain number of injections, various distinct and characteristic lesions were found. It seems, however, that not all animals are equally susceptible. What has been observed in the numerous experiments with adrenalin seems to be true also for nicotin. Now and then, how frequently the authors were not able to say, rabbits are found that will respond to the daily nicotin injection with the typical convulsion, but after months of this treatment fail to show any of the characteristic lesions about to be described. These lesions seem to be identical in every respect with those found after intravenous injections of adrenalin. After 18 injections slight changes are apparent in the bulb and arch of the aorta. After 38 injections very marked and characteristic macroscopic and microscopic lesions can be recognized. Aneurysmatic dilations of the aorta are very distinctly visible. There may be either a single aneurysm, or, what is more frequent, several in various parts of the vessel.

These dilations, as a rule, do not involve the entire circumference of the vessel, but only a limited portion of it, thus presenting the appearance of aneurysmatic pouches. On the interior surface of the aneurysmatic dilations and their immediate neighborhood, larger and smaller patches of calcification of varying shapes are

apparent. Their margin is somewhat raised above the surface of the intima, their center somewhat depressed. The more numerous the injections the more pronounced and extensive the alterations appear, but they are always of the same character. The authors have not yet concluded their experiments and they have not yet been able to carry the number of injections beyond 50. The lesions here described have nothing in common with human arteriosclerosis. They are in every essential identical with what B. Fischer describes as the result of adrenalin and digalen injections. It can be demonstrated that the primary lesion takes place in the muscle cells of the media and first of all in those nearest to the intima. Here the nuclei become broken up, the chromatin is scattered, the entire cell becomes necrotic and is finally destroyed. This process gradually extends downward in the direction of the adventitia. As the muscle cells disappear, the elastic fibers, under pressure of the blood-current, are first stretched, then broken up. The entire wall of the vessel in this spot is thus attenuated and distended and finally calcified. There is distinct *arterial necrosis*. Thus far the authors have been able to find these lesions only in the aorta. The fact that they are found mainly in the aorta, that they occur in patches, that they begin with necrosis of the muscle cells and that thus far only adrenalin, digalen and nicotin, all three vasoconstrictors, have been found to produce them, would suggest an affection of the vasovasorum as the underlying cause. This, however, is not yet proved.

In all advanced cases the left heart has been found hypertrophied. Certain minute lesions have been found in the heart muscle. The kidneys have thus far only shown a moderate degree of hyperemia. An occasional trace of albumin appeared in the urine but never any sugar. In every case that has received a sufficient number of injections very definite changes are noted in the liver. The liver cells appear entirely normal, as do also the central vein and the interlobular vessels, but the interlobular bile ducts, even at a very early period, are found surrounded by a mantle of leucocytes which increases in size after the injections are continued. The leucocytes not only surround the ducts but are found within the walls and even in the interior of the duct overlying the epithelium. This latter is always perfectly normal and the

lumen, though perhaps here and there partially obstructed by leucocytes, is always sufficiently open to permit the free passage of bile. Bile is never found in the urine. In no case have the authors ever found anything suggesting cirrhosis or degeneration of the liver cells.

18 (110). **"Tumors of wild animals under natural conditions": HARLOW BROOKS.**

The author referred to the great importance of the etiology of neoplasms and the well-recognized fact that research along this line must now rest almost entirely on experimental studies of the lower animals. By this series of observations the author hoped to establish what may be called a "normal" rate of occurrence. This can be based only on observations of large numbers of animals which have been in captivity for only relatively short periods and which must be kept under far different conditions than is possible in the ordinary zoölogical park or in the laboratory animal house.

The author's observations were made on a large number of wild animals, most of which were captured direct from the wild, and which after capture and transportation were placed under the most carefully studied natural conditions ever attempted in any large zoölogical collection.

The occurrence rate of new growths in such a group of animals, comprising most of the known species of the reptiles, birds, and mammals should furnish a valuable contribution to the study of the etiology of tumors, especially since the animals included in this collection were, for the most part, at least, pure and uncontaminated, except for such crossing as normally takes place in nature. The animals of the New York Zoölogical Society have been selected by experts for their purity of type and every one is submitted to a careful veterinary examination before becoming a member of the collection. Notwithstanding that this examination might have been expected in some cases to have excluded animals afflicted with tumors, the records show that none have been rejected for this defect.

Of 2,645 living animals which have been under the charge of the author and his associates for the past five years, no case of

true neoplasm has been found. Seven hundred and forty-four animals have died, and, as is the routine custom at the New York Zoölogical Park, have been autopsied, either by the resident pathologist or by the author. In this series of 744 consecutive cases but one case of tumor has been found. This case, significantly enough, was found in a white raccoon dog, an animal whose purity of species is decidedly in question and which has been classed by some zoölogists as a "sport" or albino. The animal has, however, been described by Hornaday as a new species, *Nyctereutes albus*. The animal was secured in northern Japan, but was unrecognized by Japanese zoölogists. The tumor in this case was found to be myxosarcoma of the ovary. Tumors of parasitic origin, granulomas, tubercles, actinomycotic foci and the like are, on the other hand, relatively common.

In addition to these data, the author also referred to various other animals, chiefly ruminants, taken in the wild, and of which none presented tumors. The latter observation was made by the author himself in the field and was in accord with statements of reliable guides and naturalists.

The author felt that the number of cases cited was sufficiently large to permit him to conclude with a reasonable amount of certainty that true neoplasms are extremely rare in wild animals living under natural conditions. Abnormal conditions of life, such as close inbreeding, semidomesticity or contamination of species as seen in dogs, horses, cattle, and particularly in those animals usually employed for laboratory experiment, notably the white mouse, unquestionably increase the relative occurrence of new growths.

19 (111). **"The cutaneous excretion of nitrogenous material":** **F. G. Benedict.**[1] (Presented by **William J. Gies.**)

A number of experiments were reported in which the subjects wore previously extracted underclothing and at the end of the experiment the nitrogenous materials were extracted with water and determined by the Kjeldahl process. Rest and work experiments were made. During rest there is considerable variation in the actual quantity of excreted nitrogen, the average of 5 experiments being

[1] *Journal of Biological Chemistry*, 1906, i, p. 263.

0.071 gm. per day. The exact nature of the nitrogenous material thus excreted was not studied. A number of experiments were made on a professional bicycler, riding a bicycle ergometer. The exercise was very severe, as the total output of heat was 600 calories per hour. The bath water and the extract water from the clothing gave a total of 0.87 gm. in a 4-hour experiment, or 0.22 gm. of nitrogen per hour.

Of greatest significance is the important bearing of this channel for the excretion of nitrogenous material in experiments on the metabolism of protein. Profuse perspiration, whether induced passively or by muscular work, results in a considerable excretion of nitrogenous material through the skin. While the work engaged in by the subjects of these experiments was severe, certainly that of some of them was not extraordinarily so, and might well be equaled by many men engaged in ordinary occupations involving muscular work. A total excretion equivalent to one or more grams of nitrogen per day is not at all inconsiderable, and hence in accurate metabolism experiments we must give recognition to the possibility of excretion through this hitherto almost unconsidered channel. Especially is this so in experiments where the total amounts of nitrogen in the ingesta and egesta are smaller than normal, since the percentage error is thereby proportionally larger.

20 (112). **"The effects of intravenous injections of solutions of dextrose upon the viscosity of the blood": RUSSELL BURTON-OPITZ.**

The experiments were performed upon dogs, in accordance with the method devised by Hürthle. When small quantities (5 c.c.) of a concentrated solution of dextrose were injected intravenously, the viscosity of the blood became slightly greater. By the administration of large quantities (50 c.c. to 100 c.c.) the viscosity was markedly decreased at first, but reassumed its normal value in the course of about one hour.

By producing artificial glycosuria, the viscosity was decidedly increased. In the latter series of experiments the surface of the pancreas was painted with solution of adrenalin. The specific gravity of the blood pursued in all cases a harmonious course with the viscosity.

Fifteenth meeting.[1]

[Third annual business meeting.]

Physiological Laboratory of the New York University and Bellevue Hospital Medical College. February 21, 1906. President Wilson in the chair.

21 (113). **"On the intermediary metabolism of lactic acid": A. R. MANDEL** and **GRAHAM LUSK.**

Administration of phlorhizin to a dog poisoned with phosphorus causes the excretion of dextrose, the mother-substance of lactic acid, and the latter then disappears from the blood and urine. On the other hand *d*-lactic acid (Kahlbaum), when given to a diabetic dog, may be completely converted into dextrose.

22 (114). **"The primary factor in thrombosis after injury to the blood-vessels": LEO LOEB.**

No uniformity of opinion exists in regard to the essential processes leading to thrombosis. According to some authors thrombosis is essentially due to coagulation of plasma or of cells. Others hold that two factors enter: Agglutination and coagulation. Klemensiewicz and Gutschy expressed the opinion that the primary formation of a fibrinous membrane at the place of injury is necessary.

We find the same diversity of views in regard to the so-called first coagulation of arthropod blood, which, as the author has already shown experimentally, is identical with thrombosis in that animal. That no explanation of thrombosis has found general recognition so far is due to the fact that microscopic examinations alone, based on staining reactions, are entirely inadequate for a decision of this question. Almost all previous work rests mainly on morphological investigation.

Sahli's work, however, forms an exception. He found that after injection of leech extract into the circulation of a rabbit, thrombi no longer formed around foreign bodies introduced into the blood-vessels. He concluded quite logically that his results prove the correctness of the view of Hanau and others, namely, that thrombosis is a process of coagulation. The results of his experiments are directly opposed to the fact repeatedly pointed out by the author, viz., that agglutination of blood plates occurs in

[1] *Science*, 1906, xxiii, p. 662; *American Medicine*, 1906, i (N. S.), p. 33.

birds and in mammalian blood outside of the body under conditions that entirely exclude coagulation, as after phosphorus poisoning, also in hirudin blood, and in bird's blood collected according to Delezenne's method.

In order to clear up these discrepancies the author carried out experiments on a relatively large number of animals. In arthropods, and especially in Limulus, he found in various experiments that the collection of blood cells around the foreign body, which leads ultimately to the formation of a hyaline thrombus, was due to a primary process of agglutination and that coagulation processes could be entirely excluded. The same applies to the extravascular coagulation of Limulus blood.

In birds and dogs the blood was made temporarily noncoagulable by injecting hirudin or peptone. In a number of experiments not only was the increase in the coagulation time of the blood in the injected animal observed, but the blood was also tested in regard to its reaction toward the tissue coagulins which accelerate the coagulation of the blood. The blood vessels were injured in different ways and were later examined microscopically. In the large majority of cases serial sections were made of each injured blood-vessel. Seven geese were used for hirudin injections, including the controls; 38 blood-vessels of 19 dogs were examined after hirudin injections (controls included); 25 blood-vessels of 12 dogs were examined after peptone injections (controls included).

The following conclusions may be drawn from the results obtained:

In invertebrates as well as vertebrates an agglutination of blood-cells or of blood-plates may take place around foreign bodies or at the place of injury of the vessel-wall. This agglutination can be present without the occurrence of any simultaneous or previous formation of fibrin. The formation of such agglutination thrombi corresponds to the clumping of the same cellular elements outside of the body, where the agglutination can take place without being accompanied by any coagulative process.

In birds the injection of hirudin does not materially alter the readiness with which a thrombus is formed. In dogs, on the other hand, it is very probable that injections of hirudin delay or may sometimes prevent the formation of agglutination thrombi. The

effect, however, is not directly due to the inhibition of the coagulation of the blood, but probably to changes in the blood which will still have to be determined.

23 (115). **"Granula and ameboid movements in the blood cells of arthropods": Leo Loeb.**

If one observes a drop of blood of Limulus, or of other arthropods, under the microscope immediately after it has left the body, an interesting phenomenon is seen. The large majority of the cell granula become smaller and soon disappear. The cells which were at first oval become round and send out hyalin protoplasm and pseudopodia. Movements of the protoplasm may be observed for a long time, but ultimately they cease, when the cells are spread out entirely and in this condition the cells gradually die.

It has been the author's aim to determine the conditions which inhibit or accelerate this apparently spontaneous dissolution of the cell granula. From the results of these investigations, which cannot be given here in detail, it follows that the fate of the granules of arthropod blood-cells depends upon certain mechanical conditions, and that the apparently spontaneous dissolution of cell granula can to a large degree be inhibited by preventing certain mechanical irritations of the cells. The changes taking place in the granules are very fine indicators of certain mechanical or chemical alterations in the environment of the cells. Such changes are determined by the character of the foreign bodies with which the cells come in contact, lipoid substances being especially favorable for the preservation of the granules. Temperature, osmotic conditions and the reaction of the medium in which the cells are suspended, influence the granules in a definite way. Furthermore, the presence of certain electrolytes is necessary for the preservation of the granules in isotonic, hypotonic, and, with the exception of sugar solutions, also in hypertonic solutions. The cell granules are dissolved in isotonic solutions of non-electrolytes. Different electrolytes exert different, specific influences.

We see, moreover, that certain substances may dissolve cell granules without enabling the protoplasm to carry out ameboid movements, but in the large majority of cases a certain parallelism is observed between the fat contents of the granules and the ameboid

movements of the cells. Whether this parallelism is due to a direct or merely to an indirect causal relation cannot at present be determined with certainty. It seems not unlikely that the ameboid movements, the spreading out of the cells and the dissolution of the granules are caused by certain metabolic changes which are induced in each instance by similar conditions. (The blood-cells of Limulus are a favorable object for demonstrating the effect of mechanical conditions upon blood cells leading to thrombosis and they can be used to advantage in courses of experimental pathology such as is given by the writer at the University of Pennsylvania.)

24 (116). **"On a course on the pathological physiology of the circulation,"** with demonstration of instruments, specimens, etc.: **W. G. MACCALLUM.**

In general in the teaching of pathology the anatomical alterations produced by disease are dwelt upon, and little attention is devoted to the detailed study of the alterations in function produced by these diseases. A course was arranged during the past year at the Johns Hopkins University to cover this ground and half of the new laboratory of experimental medicine was planned to give facilities for this work.

The aim of the course was to reproduce experimentally such diseased conditions as are seen by the students in the wards of the hospital so that they might be studied with the aid of any or all of the methods at the command of the physiologist and of the pathologist. The study of the anatomical changes which are usually found in such conditions was carried on together with these experiments.

It was planned to attempt the study of only a limited portion of the subject each year, and during the past term the diseases of the circulatory system have occupied the attention of the class. Next year it is intended to study the digestive system in a similar way.

Only those lesions were produced of which experimental study was certain to be of value — thus in the case of the pericardium, while various infections might have been used to give rise to an exudate, the blood-pressure relations, changes in heart-beat, heart-sounds, etc., were studied during the distention of the pericardium with water.

Similarly it was thought sufficient to study mechanical injuries of the heart-valves rather than to attempt their production with the aid of bacteria. Therefore, while the actual lesions were studied in the museum and histologically, the injuries to the valves were produced by cutting the valves with a special blunt hook having a knife edge on the inner side of the curve. The pressure relations were then rendered visible to the students by the curves traced in inks of different colors from cannulas inserted at various points in the circulation. Stenoses were produced by the application of a screw clamp about the orifice of the heart concerned and tracings taken in a similar way. These experiments are similar to those described by v. Basch and Moritz, but they are not subject to the criticism that they are made on a model of glass and rubber.

Murmurs could be heard and traced very accurately by the use of a stethoscope with very small bell, which could be applied directly to the ventricles or along the vessels. Thrills could be felt and the dilation and excessive activity of any portion of the circulatory apparatus directly observed. In this way there were produced and studied aortic stenosis and insufficiency, mitral stenosis and insufficiency, pulmonary stenosis, and tricuspid stenosis and insufficiency.

Lesions of the myocardium were simulated both by the mechanical destruction of the muscle substance and by the injection into it of such coagulating substances as alcohol, and the effects studied by the same method. Obstruction of various branches of the coronaries was also studied in detail.

The effect of the closure of various blood vessels was demonstrated as well as the effect of the dilation and contraction of capillaries in different regions, and the character of the capillaries of the lungs in this respect was studied. The mode of obliteration of blood-vessels after ligature and the accomodative changes which take place when the blood-supply is diminished and when collateral circulation is demanded were also considered.

The short course ended with the study of aneurysm, arteriosclerosis, and the experimental formation of thrombi and of infarcts on the introduction of foreign bodies as emboli.

The advantage which accrues to the student seems to be chiefly in his obtaining an intimate and first-hand knowledge of all the

details of processes commonly seen clinically but about which much theorizing must be done in the wards.

25 (117). **"On the blood-pressure relations in mitral insufficiency and stenosis": W. G. MACCALLUM** and **R. D. MCCLURE.**

In the course of experiments like those described in the preceding communication, blood pressure in various portions of the circulatory apparatus was recorded after mitral insufficiency had been produced by introducing a curved knife hook into the left auricular appendage and cutting some portion of the mitral valve — a systolic murmur could then be heard especially loud over the auricle and along the pulmonary veins with usually a thrill felt over the auricle. Interest attaches especially to the exact explanation of the hypertrophy of the right ventricle since, as Gerhart points out, there is an obvious obstruction to the flow of blood through the mitral orifice into the ventricle.

This is true only when the left ventricle at once accomodates itself to the condition by dilating to receive the excessive amount of blood which accumulates in the auricle, that is, the amount thrown into it from the right ventricle plus the amount regurgitated, and then succeeds in expelling it all. Unless this happens the auricle is unable to empty itself and a condition arises in which the amount of blood circulated is smaller than normal, the remainder being stagnant in the pulmonary circulation and the right ventricle is found to be driving a uniformly smaller amount of blood into a cavity (the pulmonary circulation) in which there is some stagnant blood and into which more is forced from the left ventricle during systole. The elevation of pressure from this stagnation need not be great and in the experiment where these conditions seem to prevail the pressure in the pulmonary is not much elevated. Ordinarily, however, the left ventricle dilates to receive the excessive blood, then regurgitates some and discharges nearly the normal amount into the aorta. The right ventricle then attempts to discharge into the pulmonary circulation the same large amount at the moment when the stronger left ventricle is also forcing into that cavity the amount constantly regurgitated. The pulmonary pressure is again not much elevated — not more than before but

the amount of blood in the general circulation is nearer the normal. It is quite true, as Jurgensen supposed and as Gerhart also believed, that the impulse from the left ventricle is directly felt by the right — even the pulsation of the left ventricle communicated through the imperfect valve to the left auricle is transmitted unchanged to the pulmonary arteries, just as the pulsation of the right ventricle is transmitted unchanged to the pulmonary veins. Since the right ventricle contracts simultaneously with the left this direct beat of the two ventricles against each other does probably account in part for the hypertrophy of the weaker right ventricle. It may be shown to occur by inserting a cannula into the cut end of the pulmonary artery toward the lung so as to receive the blood through the lung where it is found that on the production of mitral insufficiency the pressure in that manometer rises and the curve shows high pulsations synchronous with those of the ventricle.

Mitral stenosis was produced by means of a clamp or by a coarse suture passed through the heart and about the mitral ring. The pressure is seen to rise very high in the pulmonary circulation but because of the smaller amount of blood left to circulate there it is lowered throughout the systemic circulation.

26 (118). **"Paramecium aurelia and mutation": Gary N. Calkins.**

The ordinary species is *Paramecium caudatum;* superficially, it resembles *P. aurelia.* The latter differs from the former in smaller size, in rounded instead of attenuated posterior end, and in the possession of two instead of one micronucleus. The last is generally regarded as the most important difference between the two species. In March, 1905, a pair of conjugating *Paramecium caudatum* was isolated from a culture in an epidemic of conjugations. The ex-conjugates had all of the characteristics of *P. aurelia.* One died before many generations in culture, the other is still living and is now in the 346th generation. This one retained the characteristics of *P. aurelia* until about the 45th generation after conjugation, when it lapsed again into the *P. caudatum* form, with one micronucleus, and other characteristics of *P. caudatum.* The latter characters are still maintained.

The observation indicates one of two things. Either this is an

interesting case of mutation of species with lapse into the parent form after several generations, or the specific characteristics are inadequate and *P. caudatum* and *P. aurelia* are but variants of one species. The latter is the more reasonable hypothesis and on grounds of priority, the common forms of paramecium should be called *Paramecium aurelia.*

Physiologically the form known as *P. caudatum* is more vigorous in culture than is *P. aurelia.* During the time that the cultures were in the *P. aurelia* phase the division rate was relatively low (four divisions in five days), but soon after the change to the caudatum form the division-rate rose to two and a half divisions per day on the average for forty days, which is the highest rate on record. With this physiological difference there was a marked difference in the relative volumes of micronucleus and cell-body but no difference in the relative volumes of macronucleus and body.

27 (119). **"Experiments with some saline purgatives given subcutaneously": JOHN AUER.**

In spite of the large amount of work which has been done regarding the effect of subcutaneous and intravenous injections of saline purgatives, investigators are still in disagreement. To mention only the most recent writers, MacCallum[1] claims that "*all these salts which act as purgatives when introduced into the stomach or intestines have the same action when injected subcutaneously or intravenously.*" Eckhardt[2] on the other hand, states that "Die Mittelsalze haben bei unseren Haustieren keine abführende Wirkung" and that "Im Gegentheil wirken sie, auf diesem Wege einverleibt, häufig verstopfend." Both authors used approximately the same dose, injected the same salts subcutaneously and intravenously and yet arrived at diametrically opposite results.

In an extensive series of experiments already published, Meltzer and the author[3] have shown, among other things, that the subcutaneous injection of magnesium sulfate does not produce purgation. In view of the peculiar properties of magnesium salts the investigation was extended to some of the other saline purgatives.

[1] MacCallum, J. B.: *American Journal of Physiology*, 1904, x, p. 101.

[2] Eckhardt: Inaugural Dissertation, Giessen, 1905.

[3] Meltzer and Auer: *American Journal of Physiology*, 1905, xiv, p. 366.

In this investigation rabbits weighing about 1,500 grams were used and the salts chiefly employed were sodium sulfate and sodium phosphate. Sodium sulfate, in 4 per cent. and 25 per cent. solutions, when injected subcutaneously in 15 c.c. doses, caused no purgation in any of the experiments. Five or six hours after an injection, the feces that were passed often weighed less than five grams and were of normal consistence and form. Only rarely did the total 24-hour fecal output exceed 15 grams and the pellets were moderately hard, dry and well formed. Similar results were obtained when 4.5 per cent. sodium phosphate, in 15 c.c. doses, was injected subcutaneously. Both salts failed to cause purgation but induced a moderate degree of constipation.

The action of sodium sulfate and sodium phosphate on intestinal peristalsis was also studied. The intestines of rabbits anesthetized by morphin were observed with and without a saline bath. The subcutaneous injection of sodium sulfate and sodium phosphate caused a definite increase in the pendular motions of the small gut, especially of the duodenum. These movements, however, were not of a character to cause the evacuation of unformed feces, an impression which was confirmed by the results already reported. Increased intestinal movements and purgation are therefore by no means synonymous terms; the two may possibly even be independent of each other. Leubuscher,[1] for instance, found that 5–10 grams of sodium sulfate or magnesium sulfate injected into the stomach of rabbits produced in the majority of cases no increase in the frequency or intensity of peristalsis.

The experiments which have been briefly reported lead to the conclusions first, that the subcutaneous injection of sodium sulfate or sodium phosphate does not produce purgation in rabbits, and secondly, that the pendular movements of the small gut are moderately increased thereby.

28 (120). **"The effects of extra stimuli upon the heart in the several stages of block, together with a theory of heart-block": JOSEPH ERLANGER.** (Presented by **S. J. MELTZER.**)

This research was undertaken with the object primarily of testing the statement made by Hering that the absence of a compen-

[1] Leubuscher: *Virchow's Archiv*, 1886, civ, p. 104.

satory pause following an extra stimulation of the ventricles of the warm-blooded heart suffices to prove that the ventricles are beating independently of the auricles.

The author's experiments on the dog's heart have shown that in partial, as well as in complete heart-block, extra systoles of the ventricles are not followed by compensatory pauses. This results from the tendency for the same number of auricular beats to elapse between the extra contraction and the next following natural contraction as intervene between two natural ventricular beats in any stage of partial block. The following may be taken as an average example: If the auriculoventricular rhythm is 3:1, a ventricular extra cycle will last through any part of such auricular cycle as may have been unfinished at the moment of stimulation, plus two more auricular cycles if more than one half of the first auricular cycle was unfinished, or plus three or more auricular cycles if less than half of the first auricular cycle was unfinished.

In partial heart-block extra systoles of the auricules do not cause contractions of the ventricles excepting, occasionally, when such extra systoles fall close to the end of a ventricular cycle; and extra contractions of the ventricles never cause contractions of the auricles.

The irritability of the ventricles in partial and complete heart-block is not reduced but rather it is increased over that which obtains in the normal heart. Furthermore, in each ventricular cycle of partial and complete heart-block the irritability of the ventricles probably increases until they pass into the refractory state which develops with their contraction.

In order to determine the significance of these results, a strip of terrapin ventricle was arranged so that rhythmic stimuli as well as extra stimuli could be thrown into either end as desired. The strip was suspended over a Gaskell clamp in such a way that the impulses passing through the strip could be blocked either partially or completely at its middle. In many such experiments it was found that when the strip would beat normally, apparently, from end to end in one direction, a partial or complete block would sometimes be unmasked when the strip was made to beat in the opposite direction. Such behavior is undoubtedly due to the fact that the impulses generated in one end of the strip are more efficient

than the impulses of the other end. With a strip showing these reactions it is possible to repeat all of the phenomena that can be obtained from a mammalian heart in block produced by compression of the auriculoventricular bundle.

These facts suggest the following theory of heart-block: Clamping the auriculoventricular bundle reduces the efficiency of the cardiac impulses that reach the ventricles. With a certain degree of pressure the impulses become subminimal with respect to the irritability of the ventricles. Such an impulse therefore fails to elicit a contraction of the ventricles. The next following auricular impulse is no stronger than the preceding one, but in the interval the irritability of the ventricles has increased to the extent that the weakened auricular impulse then acts as an efficient stimulus. In this state of affairs the rhythm would be 2:1. A further reduction in the efficiency of the auricular impulse would give higher degrees of partial block and finally complete block. With this theory as a basis it becomes possible to explain all of the important phenomena of heart-block.

29 (121). **"On the nature of the reflexes controlling the successive movements in the mechanism of deglutition": S. J. Meltzer.**

The entire act of deglutition consists of a series of consecutive movements beginning with the elevation of the mylohyoid muscle of the floor of the mouth, progressing through pharynx and esophagus and terminating with the contraction of the cardia at the entrance of the stomach. The progress of these movements is surprisingly well regulated and stable. Each section of this canal enters into the peristaltic movement invariably at a given interval after the beginning of the swallowing. The time allowed for the entire course differs with each species of animal; it is about 7 seconds for the human being, about four seconds for the dog, and about 2 seconds for the rabbit.

It was early recognized that these stable relations were under the control of a reflex mechanism. That the contractions could not be caused by a direct stimulation of the muscle coat of the esophagus by the passing food was proved by the fact that there is no peristalsis when the vagi are cut. In a series of experiments

carried out by Ludwig and Wild at about the middle of the last century, it was found that ligation or transverse section of the esophagus prevents the further progress of the peristalsis to the lower segment. They drew the conclusion that the reflex is of a local nature, that is, that the food or drink while passing the esophagus sends up from each transversed section a sensory impulse which causes a reflex contraction of that section. Some twenty-five years later, however, A. Mosso made similar experiments and obtained opposite results; namely, that after ligating, transverse cutting, and even after removing a whole ring of the esophagus, the peristalsis once begun would appear also in the lower end of the esophagus. Similar observations were made by Kronecker and the author about 25 years ago on the cardia of rabbits. Even after removal of a large part of the esophagus the cardia would contract in due time after the beginning of swallowing. These experiments seem to permit only one conclusion, namely, that there are no local reflexes, that is, that the food while passing the esophagus does not send up sensory impulses to the center of deglutition, but that there is only one sensory impulse sent up at the beginning of the act of deglutition which spreads slowly within the center and sends down consecutively motor impulses to the successive sections of the deglutition path.

A few years ago the author reinvestigated the subject. There was a direct contradiction in point of fact between Ludwig and Wild on one hand, and Mosso and Kronecker and the author on the other; it seemed strange that Ludwig, the master physiologist, should have failed to see what appeared so easy to observe. An analysis of the methods employed in both series of investigations led to discovery of the reason for the discrepancy in the results. The animals of Ludwig and Wild were in deep anesthesia during the experiments, while those of Mosso were out of the anesthesia again, and the animals of the author's experiments were only under slight anesthesia. The author tested this point on a few animals and found the surmise correct. When the animals were in deep anesthesia no peristalsis passed beneath a ligature, while it ran down the entire esophagus as soon as the animals were out of the anesthesia. This means that in normal animals the process of deglutition is carried out by a reflex with only one initial sensory

impulse, the impulse traveling further within the center, while in anesthetized animals the progress of the peristalsis is furthered by a chain of local reflexes.

On reinvestigating this problem the author recently found that this chain of local reflexes exists also in the normal, non-anesthetized animals when ordinary stimulation is avoided. The experiments were made on rabbits. A cannula was tied in the upper end of the esophagus. When any indifferent liquid was injected directly into the esophagus, instead of being introduced by way of the mouth, a peristaltic wave ran down the esophagus, terminating in a contraction of the cardia just as after a normal deglutition. When the esophagus was ligated the wave stopped at the ligature. This happened whether the animal was deeply anesthetized or was awake. That the wave of peristalsis was a reflex phenomenon, and not simply due to the mechanical effect of the injections was proved by cutting the vagi. In this case the injections simply filled up the esophagus without causing any peristaltic waves or any contractions of the cardia. Better than cutting was the painting of the vagi with cocain or with magnesium sulfate. In these cases the nerves could be restored by irrigation and the experiment repeated many times. A similar wave of peristalsis was obtained when the liquid was injected through the cardia upward through a catheter. The peristalsis began at a point just above the highest drop of liquid. The wave always ran towards the stomach and against the stream, even if the animal was kept head downward. The peristaltic wave could also be produced by merely injecting air into the esophagus either from above or from below. This explains a fact which the author recorded 23 years ago, namely, that each act of "belching" is followed by a peristaltic wave of the esophagus.

The author found that this chain of local reflexes is very resistant to ether anesthesia; it disappears at about the same time that the lid reflex is abolished, and returns as soon as the ether is discontinued.

The experiments demonstrate that the function of deglutition is provided with two sets of reflex mechanisms. One mechanism has only one initial afferent impulse which travels within the center independently of any further aid from the esophagus; it is very sensitive to anesthesia and we may call it a higher reflex. The

other is a lower reflex, consisting of a chain of local reflexes which are very resistant to anesthesia.

The complexity of their mutual relations furnishes suggestive problems for future investigations.

30 (122). **"The enzymes of inflammatory exudates. A study of the enzymes concerned in inflammation and their relation to various types of phagocytic cells": EUGENE L. OPIE.**

The leucocytes of an inflammatory exudate produced by injecting aleuronat into the pleural cavities of dogs digest protein both in an alkaline and in an acid medium (uncoagulable protein nitrogen being estimated by the Kjeldahl method). The following evidence shows that two enzymes are present:

(*a*) Cells, dried after treatment with absolute alcohol and ether and then reduced to a powder, digest actively in an alkaline medium (0.2 per cent. sodium carbonate), but have almost completely lost the power of digesting in an acid medium (0.2 per cent. acetic acid).

(*b*) By subjecting washed cells of a sterile inflammatory exudate to varying degrees of heat, their power to digest in an alkaline and in an acid medium is lost at a temperature above 70° C. At temperatures between 55° and 65° C. the power to digest in an alkaline medium is unimpaired but in an acid medium it is much diminished.

(*c*) With cells of exudate removed from the pleural cavity twenty-four hours after the injection of aleuronat, digestion is very active in an alkaline medium, but less active in an acid medium. At the end of from three to five days, power of digesting in an alkaline medium is diminished or unchanged, but the acid digesting power is increased.

At the end of twenty-four hours after injection of aleuronat polynuclear leucocytes with fine granulation are predominant and from 85 to 90 per cent of the cells are present. The cells, according to observations previously reported, contain a ferment with acts in an alkaline medium. At a later stage of inflammation when large mononuclear phagocytic cells are predominant, the power of digesting in an acid medium is increased and bears a relation to the proportion of mononuclear phagocytes. If washed red

blood corpuscles of the rabbit are injected into the pleural cavity of the dog, at the end of twenty-four hours an exudate is produced very rich in large mononuclear cells, and, in correspondence, the power of the cells to digest in an acid medium is greater than that of the twenty-four hour aleuronat exudate.

Lymphatic glands in the neighborhood of the inflammatory exudate, the substernal glands in the case of the pleura, contain at the end of three or more days in greater number larger mononuclear phagocytes similar to those found in the exudate at the same stage. Emulsions made from such glands digest in an acid medium and little if at all in an alkaline medium. The digestive power of these glands (measured in cubic centimeters of 1/10 *n* sulfuric acid) is constantly greater than that of glands such as the mesenteric some distance from the seat of inflammation.

	Exp. 1.	Exp. 2.	Exp. 3.	Exp. 4.
Substernal Glands	7.6 c.c.	11.45 c.c.	11.95 c.c.	12.2 c.c.
Mesenteric Glands	—	9.9 c.c.	8.75 c.c.	9.0 c.c.
Time after injection	1 day	3 days	4 days	5 days

The differences in degree of digestion are more significant when it is recalled that the activity of other proteolytic enzymes has been shown to vary in a proportion equal approximately to the square root of the quantity of the enzyme.

The phagocytic cells of an inflammatory exudate contain two enzymes. One of these ferments, characterized by its power to digest protein in an alkaline medium, is contained in the polynuclear leucocytes with fine granulation, and since it is derived from the bone marrow may be designated *myelo-protease*. The second ferment, characterized by its power to digest only in an acid medium, in this respect resembling the autolytic ferments of other organs, is contained in the large mononuclear cells of the exudate and is increased in lymphatic glands adjacent to the seat of inflammation; it may be designated *lympho-protease*.

31 (123). **"Experimental myocarditis. A study of the histological changes following intravenous injections of adrenalin":**[1] **RICHARD M. PEARCE.** (Presented by **EUGENE L. OPIE.**)

Intravenous injections of adrenalin in doses of one-tenth cubic centimeter, soon raised to five-tenths and given on alternate days,

[1] *Journal of Experimental Medicine*, 1906, viii, p. 400.

cause, in addition to lesions of the aorta, degenerative changes in the myocardium which are most marked after the fifth injection. The majority of the animals (rabbits) which recover from the early injections exhibit a fibrous myocarditis either focal or diffuse. These proliferative changes are not analogous to those occasionally produced experimentally by bacterial toxins, but resemble rather those following obstruction of the coronary arteries. It is essentially a process of repair following degeneration of muscle fibers. The latter is due apparently to temporary ischemia of terminal vascular territories at a time when the heart muscle exerts an increased contractile effort necessary to overcome the greatly augmented intra-vascular tension. Thus both nutritive and mechanical disturbances appear to play a part in its etiology.

32 (124). **"Stable and detachable agglutinogen of typhoid bacilli": B. H. BUXTON** and **J. C. TORREY.**

By heating an emulsion of typhoid bacilli to 72° C. for half an hour a detachable agglutinin may be separated from the bacilli. This may be obtained in the filtrate on passage through a Berkefeld filter. Rabbits, which have been inoculated on the one hand by this filtrate and on the other by the heated bacilli, which have been thoroughly washed, show specific differences in their serums, as regards agglutination. The animal inoculated with the washed bacilli or stable agglutinin, produces a serum which agglutinates normal typhoid bacilli very slowly and with the formation of fine clumps. In contrast to this, the filtrate containing only detachable agglutinin gives rise to serum which clumps normal typhoid bacilli rapidly and with the formation of large flocculi.

Absorption experiments show, furthermore, that the *s* or stable agglutinin and the *d* or detachable agglutinin are distinct in character, for the heated and washed bacilli absorb nothing from the filtrate serum, but absorb all the agglutinin for normal typhoid bacilli from the bacillus serum. On the other hand the filtrate absorbs nothing from the bacillus serum, but takes up all the agglutinin from the filtrate serum.

It has also been determined that the substance in typhoid bacilli which gives rise to precipitins for filtrates of typhoid cultures is split off from the bacilli, together with the detachable agglutinums.

The possibility, further, suggests itself that the *d* agglutinin and the precipitin in a typhoid serum are identical.

33 (125). **"The effect of alcohol on hepatic glycogenesis":** **William Salant.**

In view of the current tendency to regard alcohol as a food it seemed desirable to make a study of its effect on hepatic glycogenesis, for if alcohol can replace the carbohydrates in food it might spare the carbohydrate radicals of the tissue proteins. An accumulation of glycogen in the liver after exclusive feeding with alcohol might therefore be expected. Indeed the work of Nebelthau,[1] who found 1.34 to 3.51 per cent. of glycogen in the liver of the hen after the administration of 10 c.c. per kilo of 96 per cent. alcohol on the seventh day of fasting, lends support to this view.

This suggestion was put to an experimental test. The investigation was carried out on rabbits which were fed exclusively on alcohol for periods of 4 to 6 days. Alcohol (30 or 60 per cent.) was given per os by means of a stomach tube in amounts varying between 3 to 9 c.c. per kilo daily. Control rabbits were subjected to the same preliminary treatment, but were given water instead of alcohol by stomach tube. At the expiration of 4 to 6 days the rabbits were killed under ether anesthesia and the livers examined for glycogen according to Pflüger's[2] shorter method. The amount of dextrose obtained by hydrolysis of the glycogen was determined by Allihn's method. Later in the course of the investigation, for reasons of economy of time, the amounts of copper were determined volumetrically by the iodin method instead of gravimetrically as originally recommended by Allihn.

The results at this stage of the investigation show that in rabbits fed exclusively on alcohol (10 c.c. of 30 per cent. alcohol per kilo or 12 c.c. of 60 per cent. alcohol per kilo daily for four or five days) there is no accumulation of glycogen in the liver, which shows that glycogen is not formed in the livers of rabbits when they are fed on alcohol alone. Previous to fasting or alcohol administration, these rabbits were fed on oats, hay and cabbage. As the for-

[1] Nebelthau: *Zeitschrift für Biologie*, 1892, xxviii, p. 146.

[2] Pflüger: *Archiv für die gesammte Physiologie*, 1902, xciii, p. 163.

TABLE I. — AMOUNTS OF GLYCOGEN IN THE LIVERS OF CONTROL RABBITS.

Exp. No.	Rabbit Wt.	Liver Wt.	Food Before Fasting.	Fasting Period.	Treatment During Fasting.	Hepatic Glycogen, Per cent.
1	820 gms.	22 gms.	C. H. O.[1]	4 days	Water given by Stomach Tube.	None
2	1320 "	41 "	" " "	5 "		"
5	1230 "	27 "	Carrots 3 days	5 "		0.139
6	970 "	22 "	" "	5 "		0.148
8	1370 "	42 "	" "	6 "		0.043
10	1265 "	38 "	" "	4 "		0.127
11	1470 "	53 "	" "	4 "		None

TABLE II. — AMOUNTS OF GLYCOGEN IN THE LIVERS OF RABBITS AFTER ADMINISTRATION OF ALCOHOL.

Exp. No.	Rabbit Wt.	Liver Wt.	Food Before Fasting.	Fasting Period.	Alcohol Per Kilo. Daily.	Hepatic Glycogen Per cent.
1 A	1120 gms.	48 gms.	C. H. O.[1]	4 days	10 c.c. 30 per cent.	None
2 A	1100 "	35 "	" " "	5 "	10 c.c. "	"
5 A	1100 "	44 "	Carrots 3 days	5 "	10 c.c. "	0.8
6 A	1300 "	48 "	" "	5 "	10 c.c. "	0.28
9	1280 "	47 "	" "	6 "	10 c.c. "	Trace
7 A	1500 "	53 "	" "	6 "	10 c.c. "	0.083
9 A	1270 "	43 "	" "	6 "	10 c.c. "	Trace
10 A	1470 "	53 "	" "	4 "	10 c.c. "	0.018
11 A	1350 "	54 "	" "	4 "	10 c.c. "	0.148
17 A	1800 "	66 "	Carrots 4 days	3½ "	12 c.c. 60 per cent.	None
18 A	1130 "	45 "	" "	4 "	15 c.c. "	None
19 A	800 "	35 "	C. H. O.	4 "	12 c.c. "	Trace

mation of glycogen *de novo* does not take place, under the influence of alcohol, a number of experiments were carried out to ascertain whether alcohol retards the disappearance of glycogen from the liver during fasting.

To test this point rabbits were brought up to a maximum of glycogen accumulation by feeding carrots for 3 days. Alcohol (10 c.c. of 30 per cent. per kilo) was then given in the way already stated, for 4, 5 or 6 days. As may be seen on inspecting Table II the results were negative. In one experiment only was an appreciable amount of glycogen found. In the rest of the experiments the amounts of glycogen obtained in alcohol fed rabbits were about the same as in the controls. In this connection it might be mentioned that the amount of glycogen found in rabbits killed after feeding carrots for three days varied between 4 and 7 per cent. Larger quantities of stronger alcohol were then tried. The administration of 12 to 15 c.c. of 60 per cent. alcohol per kilo daily for 4 days, after bring-

[1] C. H. O. — Cabbage, Hay, Oats.

ing the rabbits up to a maximum of glycogen accumulation by feeding carrots, was not accompanied by a retardation of the disappearance of glycogen from the liver. In the two rabbits examined the liver was glycogen free. It is safe to conclude, therefore, that alcohol when given in large amounts to healthy rabbits neither causes the formation nor retards the disappearance of glycogen from the liver.

34 (126). **"The viscosity of the blood during fever and after injection of phenylhydrazin": Russell Burton-Opitz.**

The author had previously shown that cold water and hot air baths produce an increased viscosity and warm water baths a decrease of the viscosity. In this communication the question was considered whether similar changes occur when the temperature of the body is raised by bacterial activity.

The experiments were performed upon three dogs during experimental peritonitis (*Staphyl. pyog. aureus*). The determinations were made at times when the temperature ranged from 38.7 to 39.5° C. and gave figures which were slightly above the average value of the viscosity of dog's blood. Its specific gravity, on the other hand, was invariably lower than normal, indicating thereby that, in spite of the loss in solids incurred during the inflammatory processes, the blood had retained a high viscosity.

In another series of experiments the viscosity was tested after subcutaneous injection of phenylhydrazin. The specific gravity of the blood was very low in all cases, the viscosity, on the other hand, very great. It may be regarded as proved, therefore, that these two factors need not preserve a direct relationship to one another. As in the previous work, the blood of these animals lost a large part of its solid matter but retained, nevertheless, a high viscosity.

Sixteenth meeting.[1]

Rockefeller Institute for Medical Research. April 18, 1906. President Flexner in the chair.

35 (127). "**On the digestion of gelatin**": **P. A. LEVENE** and **W. A. BEATTY.**

A complete separation of all aminoacids arising on hydrolysis of proteins was effected with the aid of phosphotungstic acid. On hydrolysis of gelatin by means of strong hydrochloric acid, glycocol, alanin, leucin, aspartic and glutamic acids, phenylalanin, prolin and oxyprolin, and a few substances of undefined nature, were obtained. On tryptic digestion a substance of the composition $C_7H_{10}N_2O_2$ was isolated. On further hydrolysis this substance yielded prolin and glycocol. The substance was evidently prolinglycyl anhydrid.

36 (128). "**The reactions of amphioxus to light**": **G. H. PARKER.**

When strong light was thrown into a basin of sea-water containing many amphioxus, the whole assembly swam about in wild confusion. This has been taken to indicate that amphioxus is very sensitive to light. But when 20 individuals were illuminated singly only 12 responded. The wild confusion in the first experiment is due quite as much to tactile stimulation as to light. When a strong, well-circumscribed beam of light was thrown on the tail of amphioxus the animal almost always reacted by a slight forward spring. When the light was thrown on the middle of the body there was usually no reaction, though sometimes a backward movement. When the light was applied to the head end, there was always a backward spring. This sensitiveness was not lost or impaired by cutting off the anterior end, including the so-called eye-spot. When cut into halves amphioxus retained sensitiveness to light in the anterior half, but not in the posterior half, though the latter was normally reactive to stimulation from very weak acid. This indicates that though amphioxus is without a brain proper, the anterior portion of its medullary tube is related to the posterior portion somewhat as the brain and cord are in the higher vertebrates. The distribution of the sensitiveness of amphioxus to light

[1] *Science*, 1906, xxiii, p. 846; *American Medicine*, 1906, i (N. S.), p. 152.

corresponds to the distribution of the "light" cells (Hesse) in its medullary tube and is probably not connected with the skin. Specimens of amphioxus tend to collect in the darker parts of an aquarium. They also swim away from a source of light. Amphioxus is therefore negatively photodynamic and negatively phototropic.

37 (129). **"The relation of blood platelets to thrombus formation": J. H. PRATT.**

In the frog, rabbit and dog experimental thrombi three to ten minutes old were studied. In the youngest thrombi there was agglutination of blood platelets or spindle cells and agglutination of erythrocytes without evidence of fibrin formation. The fusing and distortion of the erythrocytes were marked. The erythrocytes were sometimes broken up into small granular masses which simulated blood plates. By the use of a sodium metaphosphate solution it was possible to distinguish the blood platelets from the degeneration products of the erythrocytes.

38 (130). **"Conditions of bacterial activity in the intestine in cases of advanced, apparently primary, anemias": C. A. HERTER.**

The author reported results of the coördinated studies of 15 cases of apparently primary advanced anemias, in ten of which the blood picture was that of pernicious anemia. The studies related to the occurrence of phenol in the urine and in the feces; of indol in the feces and indican in the urine; of skatol in the feces; to the Ehrlich aldehyde reaction of the urine; to the Ehrlich aldehyde reaction of the feces; and to the hydrobilirubin reaction of Schmidt. In the case of indol, phenol and skatol, quantitative studies were made. The observations established the fact that in so-called primary, pernicious and allied anemias the indications of excessive putrefactive decomposition are almost regularly pronounced. These changes are associated with definite and characteristic departures in the bacterial activity of the intestinal flora studied in fermentation tube experiments. A careful study of the microscopic fecal fields, of the sedimentary fields in fermentation tubes, of the anerobic plates from the sterilized feces, and of the results of a modification of Welch's incubation test for the gas-bacillus, indicates that

in nearly every instance examined the peculiar Sacchus-butyric type of bacterial decomposition here found is dependent upon *B. welchii* (*B. ærogenes capsulatus*). Evidence is furthermore brought forward to show that this organism is a prominent and perhaps specific factor in some cases of advanced "primary" anemia. The overgrowth of the gas-bacillus is associated with a partial disappearance of *B. coli*. During convalescence the gas-bacillus recedes numerically and *B. coli* resumes a dominant position.

39 (131). **"Absorption of typhoid bacilli from the peritoneal cavity"**: **B. H. BUXTON** and **J. C. TORREY**.

Shortly after injection of typhoid bacilli into the peritoneal cavity of a rabbit the organs in most experiments are found to be invaded by the bacilli, more particularly the liver and spleen, in which there may be enormous numbers. By means of injection of lamp black, the peritoneal path for this rapid rush to the organ is shown to be by way of the anterior mediastinal lymphatic trunks. Even in five minutes after injection the trunks and the anterior mediastinal lymph node are markedly blackened.

On plating out the lymph nodes after injection of typhoid bacilli, they are often found to contain many millions of bacilli, and, as a general rule, if there are many bacilli in the lymph nodes there are also many in the organs.

40 (132). **"The dicrotic elevation at different points of the arterial tree"**: **PERCY M. DAWSON**. (Presented by **J. R. MURLIN**.)

In a number of dogs the form of the pulse-wave was studied by means of the Hürthle manometer. The arteries upon which the observations were made were the following—aorta, brachiocephalic, innominate, carotids, thyroids, vertebrals, internal mammaries, axillaries, brachials, left subclavian, celiac axis, superior mesenteric, left renal, inferior mesenteric, left iliac, deep femoral, femoral, saphenous and peripheral end of the carotid, *i. e.*, a side branch of the circle of Willis. The exact values of the apex and base of the pressure triangles were determined from readings of the systolic and diastolic pressures obtained by means of a valved manometer.

A careful study and comparison of the results has led to the following conclusions.

1. In passing from the heart to the periphery the dicrotic elevation increases in distinctness and in the special case of the aorticofemoral system the dicrotic elevation occurs lower on the catacrotic limb of the fundamental wave. On the other hand as the arteries decrease in size, the dicrotic elevation soon disappears, *e. g.*, in the thyroid, saphenous and so forth. Consequently there is in every system of arteries (aorticofemoral, brachiocephalic and left subclavian) a region lying somewhere between the aortic arch and the periphery in which the dicrotic elevation is maximal.

2. In the aorticofemoral system the side pulse shows a maximal dicrotic wave between the origin of the renal and that of the deep femoral artery; in the brachiocephalic system, between the origin of the carotids and that of the vertebral or thyroid artery; in the left subclavian system the dicrotic wave is less pronounced in the mammary than in the vertebral artery and consequently the maximum in question must lie central from the origin of the former artery. In the case of the end pulse, the region of the maximal dicrotic wave is in or peripheral to the brachial, femoral and carotid arteries but it is impossible to say whether the maximum occurs in them or peripheral to them, because they were the most peripheral of the arteries examined in this connection.

3. In the femoral pulse wave the dicrotic elevation is normally much more distinctly marked and begins much lower on the catacrotic limb of the fundamental wave than is the case with the carotid pulse.

4. Certain operative procedures (namely determination of the blood pressures in various deep seated arteries after opening the thoracic or abdominal cavity) cause the predicrotic notch in the femoral to become more and more pronounced so that ultimately the dicrotic wave appears as an elevation on the ascending limb of the fundamental wave which immediately follows. In the case of the carotid pulse this effect of operation is very rarely seen. As yet the writer is unable to offer any satisfactory explanation of these local variations in the character of the dicrotic elevation. He has however begun an investigation with the view of elucidating this question.

41 (133). **"The Influence of Subcutaneous Injections of Dextrose upon Nitrogenous Metabolism": FRANK P. UNDERHILL** and **OLIVER E. CLOSSON.** (Presented by **WILLIAM J. GIES.**)

It has been frequently assumed that the large quantity of sugar present in the body in the condition of diabetes is responsible in part for some of the characteristic symptoms noted. For example, it has been asserted that various acids or acid derivatives may be formed giving rise to the condition of acidosis, as indicated by the well-known increased output of ammonia by diabetics. What influence the large quantity of sugar may have upon the distribution of nitrogen in the urine has received but scanty attention, especially with accurate methods.

Recently Scott (J. Physiol., 18, p. 107) has attempted to imitate the condition which obtains in diabetes by injecting into dogs large quantities (seven grams per kilo) of dextrose subcutaneously, and has made a study of the distribution of the urea, non-urea, and ammonia nitrogen as compared with the distribution in the normal animal. He has shown that when the above mentioned quantity of dextrose is injected there is an increased protein metabolism. Further there is probably excreted an increased output of ammonia combined with an acid or acids derived from the decomposition of the dextrose. There is also a diminution in the proportion of nitrogen eliminated as urea and an increase in the output of the non-urea nitrogen.

It was the purpose of the present investigation to study the character of this non-urea nitrogen. Accordingly the total nitrogen, urea nitrogen, ammonia nitrogen, creatinin nitrogen, uric-acid nitrogen, and purin nitrogen have been determined under conditions similar to those of Scott's experiments. In harmony with Scott's results, the authors found an increase in the total output of nitrogen due to increased metabolism, together with an increased elimination of oxalic acid. In no case, however, did they observe a significant change in the proportions of the various forms of excreted nitrogen.

The discrepancies between the two series of results can be accounted for in part by the fact that most of Scott's dogs were suffering from severe cystitis due to catheterization. It is well known that cystitis is sufficient to give rise to an increased excre-

tion of ammonia at the expense of the urea. It is therefore concluded that subcutaneous injections of large quantities of dextrose do not give any evidence of toxic action, that is, of an acidosis, as advocated by Scott.

The experiments suggest that the subcutaneous injection of large quantities of dextrose may be useful as a method of parenteral feeding, since quantities up to seven grams per kilo in the dog and rabbit may be given without the appearance in the urine of more than the merest trace of the sugar.

42 (134). **"Diffusion into Colloids and a Biological Method for Testing the Rate of Diffusion": SIMON FLEXNER** and **HIDEYO NOGUCHI.**

Certain experiments on the destructive action of bile and bile salts upon the pancreas made by Flexner indicated that colloidal substances restrained the action of the salts. It was suggested that this restraint probably depended upon a reduction in the rapidity of diffusion of the salts into the tissues. The studies of Voigtländer on the influence of colloids (agar-agar) on the rate of diffusion of certain crystalloids tended to show that diffusion into agar-agar jelly takes place at about the same rate as into water. The experiments summarized in this communication were made with hemolytic substances suspended in isotonic saline solution and in agar-agar and gelatin jelly. The rate of diffusion could be measured by the depth and degree of hemolysis produced in a jelly containing in suspension susceptible red blood-corpuscles. The experiments were varied. The red corpuscles were suspended in the warm jelly which was permitted to congeal. The blood jelly was overlaid with the hemolyzing agent dissolved in saline solution, or this agent was also contained in a solidified jelly. The hemolyzer was made to diffuse either downwards or upwards according as the blood, or hemolyzer, jelly was above or below. Moreover, the hemolyzer was placed in the jelly and made to diffuse upwards into a watery solution, the amount of diffusion being measured by the degree of hemolysis caused by the fluid removed at given intervals. Two factors were always considered, extent or degree of hemolysis, and time.

The substances employed were mineral and organic acids, alkalies, sodium taurocholate, saponin, solanin, venom, and tetanolysin. The results can be stated in general terms as follows:

Acids, alkalies, salts, glucosids, and toxin diffuse into 0.9 per cent. watery NaCl solution more quickly than into a similar solution containing agar-agar and gelatin. This reduction in rapidity of diffusion increases with increase in concentration of the jelly. Ten per cent. gelatin exerts a greater inhibition than two per cent. agar-agar, and 25 per cent. gelatin exerts greater restraint than 10 per cent. gelatin. The ratio between the rate of diffusion and the concentration of the colloidal suspension is, in the case of gelatin, nearly inversely proportional to the square root of the concentration of the colloid. In the case of agar-agar, with which the possibility of varying the concentration is far less than with gelatin, the inhibitory influence is less marked and does not conform to this rule. Voigtländer's results are applicable to the special case of agar-agar jelly.

The influence of colloids upon the injurious effects produced by bile salts upon the pancreas is due, apparently, to a modification by reduction of the diffusibility of the bile salts, which result diminishes the concentration of the salts brought in contact with the pancreatic tissues in a unit of time.

Seventeenth meeting.[1]

Laboratory of the Department of Health, of New York (East 16th St.). May 23, 1906. President Flexner in the chair.

43 (135). **"Analogies between the phosphorized fats obtained from the brain and kidney,"** with exhibition of products: **EDWARD K. DUNHAM.**

So much attention has been directed to the protein constituents of protoplasm that it has become usual to regard proteins as the physical basis of life. Relatively recent investigations have, however, indicated that all cells contain complex substances of a fatty or lipoid nature, in which phosphorus and nitrogen are conspicuous elements. Many of these lipoids possess remarkable physical properties. In contact with water or alkaline liquids, they pass into colloidal solution after imbibing large quantities of water with the production of "myelin forms." They also differ from neutral fats in doubly refracting light. Such physical characters and the complex molecular constitution of these lipoids appear to justify the assumption that they, as well as proteins, are essential con-

[1] *Science*, 1906, xxiii, p. 979; *American Medicine*, 1906, i (N. S.), p. 155.

stituents of protoplasm and that a study of living matter must include the consideration of these compounds of the fatty acids.

The phosphorized fats, or lipoids, which have been most carefully studied have been obtained from the brain,[1] but even as derived from this source, where they are believed to be present in relatively large amounts, their constitution and mutual relationships have not been clearly established.

During investigations of alcoholic extracts of kidneys, the writer has been led to infer that substances closely related to the lipoids derived from the brain may be obtained by similar methods from the kidney, and the purpose of the present communication is to report a few representative analyses from among those upon which the inference just stated is based.

Extracts of finely divided renal tissue, freed from obvious fat, made with hot 85 per cent. alcohol, yield a precipitate, upon cooling, which contains a variety of lipoids, while certain others remain in solution. For convenience, those lipoids which are relatively insoluble in cold alcohol may be classed as the "protagon" group, and those not precipitated on chilling as the "lecithin" group. A preliminary purification of the "protagon" group was effected by treating the crude precipitate with benzol, which left a small residue undissolved. From the concentrated solution in benzol a powdery precipitate was formed upon the addition of a mixture of acetone and rhigolene and became pure white when repeatedly washed with the latter mixture, in which it was nearly if not wholly insoluble. This precipitate was soluble in hot 85 per cent. alcohol, from which it separated in discoid crystals on cooling the solution. It corresponded in solubilities to Liebreich's "protagon" or to an impure sphingomyelin described by Thudichum, and obtained from the brain. It contained 2.869 per cent. of phosphorus and 3.126 per cent. of nitrogen. A portion of this precipitate was dissolved in hot 85 per cent. alcohol and an alcoholic solution of lead acetate was added to excess. The mixture was boiled and filtered while hot. Upon cooling, a heavy white crystalline precipitate formed. This was removed by filtration and recrystallized from 85 per cent. alcohol four times. The

[1] Thudichum: Die Chemische Konstitution des Gehirns des Menschen und der Tiere. Tübingen, 1901.

white crystalline powder so obtained was analyzed. (See Table I, 160 A.) A second sample of the same substance, prepared by the same method from another lot of kidneys, but recrystallized only once, and therefore, which was less pure, contained nearly the same percentages of phosphorus and nitrogen, allowance being made, in the calculations, for the 1.77 per cent. of lead in it (Table I, 160 B).

The solubilities and reactions of this substance correspond to those of the compound which Thudichum calls "sphingomyelin," when it contains, as impurities, small quantities of kerasin and a cerebroside to which he assigned no name. Upon hydrolysis with barium hydrate, this substance from the kidney yields ammonia, trimethylamin, a substance reducing Fehling's solution, and, apparently, an acid forming a barium salt which is insoluble in a mixture of absolute alcohol and ether. These cleavages are analogous to those observed by Thudichum on hydrolysis of his sphingomyelin.

A portion of the lead-free substance (160 A) was dissolved in hot 85 per cent. alcohol and precipitated with cadmium chlorid in alcoholic solution. The precipitate was removed by filtration, redissolved in 85 per cent. alcohol and kept at 30° C. over night. This procedure separates kerasin from sphingomyelin. The precipitate that had formed during the night was rapidly removed with a Buchner filter, pressed, dried and analyzed (Table I, 160 A—$CdCl_2$). The percentages of phosphorus and nitrogen in a second but less pure sample of this cadmium chlorid compound are also given (Table I, 160 B—$CdCl_2$), and, for comparison with these, the results of analyses of "sphingomyelin" and "apomyelin" by Thudichum. He obtained cadmium salts containing 16.86 per cent. $CdCl_2$ and 26.59 per cent. $CdCl_2$, and believed that these variations depend upon the relative abundance of compounds containing one and two molecules of cadmium chlorid, respectively; assigning the limits 16.4 per cent. $CdCl_2$ and 28 per cent. $CdCl_2$ to these two hypothetical compounds. The percentages of cadmium chlorid found in the similar products from the kidney fall within these limits, and the percentages of cadmium and chlorin are in close accord with the assumption that the cadmium is present as chlorid, thus indicating that the salt is an addition product.

The acetone rhigolene mixture used in the preliminary purification of the "protagon" group contains substances which possess solubilities similar to those of Thudichum's kephalin and myelin, both before and after precipitation with lead acetate. But these substances have not yet been obtained in sufficient quantities for satisfactory purification. They appear, however, to contain more phosphorus (over 4 per cent.) and less nitrogen (about 1 per cent.) than the analogue of sphingomyelin already considered, and these characters are also in harmony with Thudichum's analyses of kephalin, myelin and sphingomyelin.

From the "lecithin" group, a cadmium chlorid compound was obtained which, when purified with ether, acetone and alcohol, resembled the cadmium chlorid compound of paramyelin (Thudichum). The percentages of phosphorus and nitrogen in this compound (150 A—$CdCl_2$), in a second sample purified but once with ether and acetone (150 B—$CdCl_2$), and in Thudichum's paramyelin from human and ox brains, are given in Table II.

Still other compounds containing phosphorus, nitrogen and fatty acids have been obtained from renal extracts, and appear to be analogous to substances derived from the brain. But since all these compounds require much further study before their constitution, relations to each other and to substances originally present in the tissues can be understood, it is believed that this preliminary report need not be burdened with further, necessarily incomplete analytical data.

Table I.

	160 A.	160 B.	160 A—$CdCl_2$	160 B—$CdCl_2$	Sphingomyelin.[1]	Apomyelin.
P	2.493	2.48	3.690	3.623	3.24	3.23
N	2.869	2.74	2.896	3.211	2.96	3.00
C	63.570	—	67.546	—	65.37	67.01
H	11.840	—	12.445	—	11.29	11.35
$CdCl_2$	—	—	25.380	25.362	16.63 to	26.59

Table II.

	150 A—$CdCl_2$	150 B—$CdCl_2$	Paramyelin Cadmium Chlorid.[2]	
			Human Brain.	Ox Brain.
P	4.348	4.156	4.78	4.313
N	2.403	2.219	2.25	2.029
$CdCl_2$	25.990	24.340	24.95	21.275

[1] Thudichum, l. c., p. 170.

[2] Thudichum, l. c., p. 153.

44 (136). "**The toxicity of indol**": **A. N. RICHARDS** and **JOHN HOWLAND.**

Previous observers have shown the comparatively slight toxicity of indol, large amounts having been given to dogs with no resulting symptoms.

A series of experiments on rats, guinea pigs and rabbits have shown that if the capacity of the cells of utilizing oxygen is diminished, as by potassium cyanid, or chloroform, the intensity and duration of symptoms following the injection of definite doses of either indol or phenol are increased.

Experiments on dogs have shown that if potassium cyanid is given together with indol (0.25–0.5 gm.) by subcutaneous injection, a series of symptoms results which ends after a period of days with the death of the animal. The symptoms consist of stupor and delirium, loss of power over limbs, exaggerated reflexes with spasticity of hind limbs, hypersensitiveness in the lumbar region, especially the tail, loss of sight, constant nausea, feces diminished in amount and bloody, and emaciation. Autopsy showed marked congestion of the mucosa of the duodenum, ileum, and colon, blood in the intestinal contents, degenerative changes in the liver and intestinal mucosa, excessive cerebrospinal fluid, and softening of the brain-tissue.

Comparable results have been obtained when prolonged chloroform anesthesia or prolonged asphyxia has been substituted for the cyanid.

In one experiment an intestinal fistula of the Thiry-Vella type was established in a dog and complete recovery from the operation allowed. On poisoning with potassium cyanid and indol, the latter could not be detected in the urine but was found in the contents of the isolated intestinal loop. Urinary examinations in the various experiments showed that diminished oxidation lessens the intensity and prolongs the duration of the indican reaction in the urine.

The experiments were made as a part of a study of the etiologic factors in recurrent vomiting in children. At the beginning of these seizures there are signs of diminished oxidation (increased elimination of uric acid, neutral sulfur, lactic acid, acetone bodies) and an abnormally intense indican reaction. It is believed that failure to oxidize completely substances of the type of indol, may

result in the production of distinct mental symptoms and in the partial excretion of the substances into the gastrointestinal tract. The disturbance induced by such substances is capable of producing nausea and vomiting.

45 (137). **"The formation of urea": L. B. Stookey** and **A. S. Granger.** (Presented by **R. A. Hatcher.**)

Subcutaneous injection of liver-extracts (dog) was found to lead, in the dog, to an increased elaboration of nitrogenous end-products into urea. Liver extracts which had been heated to 55° C. failed to manifest this stimulative action. These results might indicate an enzymatic formation of urea. Further experiments are in progress.

46 (138). **"The effects on embryonic development of the Röntgen rays acting on the spermatozoa of the toad previous to fertilization": C. R. Bardeen.** (Presented by **Eugene L. Opie.**)

Experiments have shown that spermatogenesis may be inhibited by exposure to the Röntgen rays or to radium. The direct action of the rays on the spermatozoa has not, apparently, been studied. It occurred to the author that it would be interesting to see if spermatozoa could be injured by the Röntgen rays and, if so, what the effect would be on the development of ova fertilized by spermatozoa thus affected. During the short breeding season of the toads in the vicinity of Madison, Wisconsin, the author collected daily several pairs of toads, separated the males from the females, and from the males got enough sperm to make a slightly cloudy suspension in water. This suspension was divided into two parts, one of which was kept for control purposes, while the other was exposed for from an hour and a half to two hours and a half to Röntgen rays. Several of the females were then opened, until, when possible, one was found in which the eggs seemed abundant and ready to be discharged. Two short strings of eggs were removed and each string was divided into two parts; one part was placed in the control dish, the other in that which had been exposed to the rays. After about fifteen minutes each string was placed in a separate dish of water.

Several of the experiments proved of negative value either be-

cause not even the spermatozoa of the control dish proved capable of fertilizing the eggs, owing to the time which had elapsed since the removal of the sperm from the male, or because none of the females happened to have ova in the right condition to be fertilized. The thoroughly successful experiments, owing to the short season of mating, were few in number but they were convincingly positive. All eggs fertilized by the control spermatozoa developed normally. Only one egg in fifty to a hundred that were fertilized by the exposed spermatozoa, developed at all normally. All the others showed marked defects in development.

The results of the experiments may be briefly summarized as follows:

1. The spermatozoa of the common toad retain power of movement and fertilization for from one-half to nearly three hours in a dish of lake water at room temperature. On hot days they die sooner than on cool days.

2. Spermatozoa when under exposure of Röntgen rays die sooner than when not thus exposed.

3. When spermatozoa are exposed to the rays so long that very few are capable of fertilizing ova, the eggs thus fertilized usually do not develop into larvas but they may do so.

4. When spermatozoa have been exposed for a considerable period to the Röntgen rays and yet are still capable of fertilizing a considerable proportion of eggs placed in the same dish the eggs seem to develop normally at first, but beyond the gastrula stage the development becomes retarded and the resulting larvas are markedly deformed. These deformities are quite varied. In one larva for instance, a considerable part of the central nervous system and the gills were undeveloped on one side while the abdominal viscera were developed only on that side. In another the central nervous system was abnormal on both sides and the alimentary canal quite defective. Considerable further study is necessary to determine accurately the nature of all the abnormalities present in the various monsters the author has preserved. Apparently all are defect abnormalities.

From the results obtained it may be concluded:

1. That nuclear material may be so influenced by exposure to the Röntgen rays that after a latent period it will call forth marked abnormalities in development.

2. That injury to spermatozoa capable of fertilizing ova may cause the development of monsters from the ova thus fertilized.

47 (139). **"A vago-esophageal reflex":** **S. J. Meltzer** and **John Auer.**

The general knowledge of the contractions of the esophagus is confined to the peristaltic movements, that is, the consecutive contractions of the successive parts of the esophagus following a normal deglutition, or, as it was described by Meltzer at a previous meeting of this society, after an injection of liquid or insufflation of air directly into the esophagus. A simultaneous contraction of the entire esophagus can be produced only by stimulating the peripheral end of the vagus when cut in the neck.

The authors discovered that in dogs a tetanic contraction of the entire esophagus can be caused also by reflex ways. When the vagus is cut in any part of the neck, an electric stimulation of its central end causes a prompt longitudinal and circular contraction of the entire esophagus, which lasts as long as the stimulation continues. Particulars and other interesting facts connected with this observation will be reported later.

48 (140). **"Ion protein compounds,"** with exhibition of products: **William J. Gies.**

About five years ago the author found that "when the electric current is passed through neutral or alkaline mucoid solutions (consisting of sodium or calcium salts of mucoids) turbidity results within a short time, and flocks eventually form and can be filtered off." This observation was included in a preliminary report of work then in progress.[1] About the same time Huiskamp had been making similar observations in connection with salts of nucleoprotein from thymus.[2] Shortly afterward, in preparing material for work in another connection,[3] the author precipitated from an alkaline solution (Na_2CO_3) of mucoid, with the aid of acetone

[1] Mead and Gies: *American Journal of Physiology*, 1902, vi (*Proc. Amer. Physiol. Soc.*, 1901, p. xxviii); also Gies and collaborators: Biochemical Researches, 1903, i, p. 53.

[2] Huiskamp: *Zeitschrift für physiologische Chemie*, 1901–'02, xxxiv, p. 32.

[3] Gies: *Loc. cit.*, 1903, viii (*Proc. Amer. Physiol. Soc.*, 1902, p. xliii); Biochemical Researches, p. 54.

after failure with alcohol, a water-soluble compound — apparently sodium mucoid. This fact has not been published hitherto, although it was stated at that time that organic compounds, such as gelato-mucoid, had been obtained.[1]

The author has lately prepared calcium, sodium, potassium and ammonium salts of mucoid by the following process: The gluco-protein was obtained in slightly alkaline solution. This solution was dialyzed until neutral and then was poured into a large excess of 95 per cent. alcohol, by which treatment the mucoid was immediately precipitated. Initial purification was effected by resolution, dialysis and reprecipitation. The products were rendered anhydrous by treatment with absolute alcohol and ether. Probably all bases yield such salts, although the author confined his remarks to salts of inorganic hydroxids.

The comparatively pure inorganic salts of the mucoids thus prepared are light, snow-white powders. They dissolve in water very readily and are dissociable products. The concentrated solutions resemble mucus. The aqueous solutions are neutral to litmus and acid to phenolphthalein. Ammonium compounds have been prepared that were acid to litmus also. The calcium salt yields about 12 per cent. of ash, whereas the corresponding mucoid is practically ash free. It is very probable that the mucins in the secretions occur in the form of such salts, as Müller has already suggested. Yeast nucleoprotein has yielded similar products. Presumably other nucleoproteins will do so also.

The author believes these observations clear the way for important discoveries connected with the glucoproteins, nucleoproteins, proteinates and similar protein products. Numerous studies in this connection were suggested in the oral communication to the Society and are proceeding with the coöperation of the workers in the author's laboratory. The best method of preparing the compounds referred to has not yet been definitely ascertained, but the author hopes to describe it in detail at an early date.

[1] Gies: Loc. cit., 1903, viii (*Proc. Amer. Physiol. Soc.*, 1902, p. xliii); *Biochemical Researches*, 1903, i, p. 54. Also Posner and Gies, *American Journal of Physiology*, 1904, xi, p. 404.

49 (141). "**Some facts showing that the brain educts termed phrenosin (1874) and cerebron (1900) were practically the same**": **WILLIAM J. GIES.**

In a discussion of the chemical heterogeneity of protagon, the author previously alluded to the probability that phrenosin and cerebron were identical.[1] Thierfelder recently published in rejoinder some opinions to the contrary.[2] Reëxamination of the facts in the case have convinced the writer that Thudichum's phrenosin, Gamgee's pseudocerebrin, Parcus's cerebrin, Kossel and Freytag's cerebrin, Thierfelder's cerebron and Koch's cerebrin were practically the same. The slight discrepancies among the figures for percentage elementary composition were probably due to slight proportions of inevitable impurities in each preparation.

Of the products referred to, phrenosin and cerebron have been subjected to the most thorough study. The descriptions of each are in close harmony. Each has been found to yield, on hydrolysis with dilute sulfuric acid, apparently the same proportions of a sugar (galactose), a nitrogenous base (sphingosin) and a peculiar organic acid. The following data were obtained on direct analysis of the latter product:

	C.	H.
Neurostearic acid (obtained by Thudichum from phrenosin)	75.94	12.64
Cerebronic acid (obtained by Thierfelder from cerebron)	75.33	12.50

If the inevitable impurities in each product are disregarded it seems obvious that the names refer to the same substance.

The following formulas were assigned to it:

Thudichum's product (Neurostearic acid)	$C_{18}H_{36}O_2$
Thierfelder's product (Cerebronic acid)	$C_{25}H_{50}O_3$

It is apparent, however, that the formulas are practically interchangeable. Each is empirically an approximate multiple of $C_8H_{17}O$. With this fact in mind the following equations that were given to represents the relations of the cleavage products to the mother substance, emphasize the opinion that phrenosin and cere-

[1] Posner and Gies: *Journal of Biological Chemistry*, 1905, i, p. 59.

[2] Thierfelder: *Zeitschrift für physiologische Chemie*, 1906, xlvi, p. 518.

bron were the same :

$$\underset{\text{Phrenosin}}{C_{41}H_{79}NO_8} + 2H_2O = \underset{\text{Neurostearic acid}}{C_{18}H_{36}O_2} + \underset{\text{Sphingosin}}{C_{17}H_{35}NO_2} + \underset{\text{Galactose}}{C_6H_{12}O_6}$$

$$\underset{\text{Cerebron}}{C_{49}H_{93}NO_9} + 2H_2O = \underset{\text{Cerebronic acid}}{C_{25}H_{50}O_3} + \underset{\text{Sphingosin}}{C_{17}H_{35}NO_2} + \underset{\text{Galactose}}{C_6H_{12}O}$$

50 (142). "**A simple electrical annunciator for use in metabolism experiments, and in connection with filtration, distillation and similar operations,**" with demonstrations : **WILLIAM H. WELKER.** (Communicated by **WILLIAM J. GIES.**)

In the paper describing his cage for metabolism experiments the writer [1] referred to the advantages of the "sliding shelf" devised as a holder for the urine receiver, and, in that connection, made the following remark : "The shelf also favors the use of electrical apparatus to ring out the time of elimination of urine-fractions, in experiments in which fractions of the urine must be examined separately and immediately after their natural excretion" (page 407). This remark alludes to one of the several additional devices the writer had intended to perfect for use with the cage described.

In order that an annunciater might be of the greatest service in metabolism work in the way already indicated, and also to insure its usefulness for filtration, distillation and other operations in which the weight of a product above a certain maximum amount could be relied upon to close an electrical circuit and announce the delivery of the material, it was necessary that it should be delicately responsive to the weight of several grams and yet be readily adjustable within relatively wide limits in that respect ; that it should be light in weight, of small compass but durable, and resistant to derangement from any cause ; also that it should hold, without risk of loss or modification of the contents, any suitable vessel placed upon it.

At the writer's request, Mr. Welker, who has exhibited in this laboratory unusual proficiency in handling electrical apparatus, devised an annunciator to meet these requirements and has perfected an apparatus that is eminently satisfactory for all the purposes contemplated.

The annunciator shown to the Society consists of two square boards ($4\frac{1}{2} \times 4\frac{1}{2} \times \frac{3}{8}$ inch) securely fastened together with a piano hinge on one side, and kept apart, by a spring perpendicularly

[1] Gies : *American Journal of Physiology*, 1905, xiv, p. 403.

arranged at the opposite side, in such a way as to permit a definite pressure to force the surface of the boards together. The spring can be adjusted so as to increase or decrease, within considerable limits of weight, the amount of force (weight) required to bring the board surfaces in contact. In the opposed surfaces of the boards platinum electrodes (plate and points) are so placed that perfect contact between them is effected when the boards are brought together and the circuit is closed. The electrodes connect with binding posts on the hinged side. A small dry cell is used. The entire apparatus, including bell attachment, may be placed on a surface 5 x 8¼ inches. The bell employed directly with the apparatus is a small one with delicate musical sound. Its ringing under a cage during a metabolism experiment does not disturb the animal. It is obvious, of course, that the apparatus can be connected with a bell in a distant room.

In the demonstration it was shown that the apparatus announced the deposit, in an ordinary urinary receiver placed on it, of volumes of water less than 5 c.c. The apparatus may be adjusted to announce a volume as small as 1 c.c. and may be made, in larger sizes, to announce the deposit of masses of any desired weight.

Various details of description that would show the particular value of the apparatus in other respects will be given in the paper soon to be published by Mr. Welker.

51 (143). **"Some observations on the presence of albumin in the bile": WILLIAM SALANT.**

The presence of albumin in the bile under pathological conditions has been noticed by several observers. Thus, Lehmann,[1] who examined post-mortem bile from the gall bladder in 100 cases, found albumin in nutmeg liver, fatty liver, and parenchymatous hepatitis. Pouchet[2] found albumin in the bile of six patients that died of cholera. Among recent observers may be mentioned Brauer[3] who has reported similar findings in typhus and parenchymatous nephritis. Hallauer[4] analyzed the bile in a number of

[1] Lehmann: *Centralblatt für die medicinischen Wissenschaften*, 1867, v, p. 172.

[2] Pouchet: *Comptes Rendus*, 1884, xcix, p. 847, also 1885, c, p. 220.

[3] Brauer: *Zeitschrift für physiologische Chemie*, 1903–'04, xl, p. 182.

[4] Hallauer: *Verhandlungen der medicinisch-physikalischen Gesellschaft*, Würzburg, 1904, p. 186.

cases. He found albumin in 5 out of 6 cases of cloudy swelling of the liver associated with pneumonia, miliary tuberculosis and sepsis; also in some cases of fatty liver, but none in cirrhosis of the liver. Experimentally, in rabbits, he obtained an albuminocholia after intravenous injection of albumose.

Within the past two years several other investigations on the experimental production of albuminocholia have been described. Brauer[1] in his paper "on the study of the liver," in the *Zeitschrift für physiologische Chemie*, reported the presence of albumin in the bile of a dog with a permanent biliary fistula after poisoning with ethyl alcohol and small quantities, 3–5 c.c., of amyl alcohol. At his suggestion Pilzecker[2] carried out a similar study on the bile of dogs with permanent gall bladder fistulas after poisoning with phosphorus and arsenic. His result seemed to corroborate the work of Brauer. Another interesting statement which both observers made was to the effect that albumin passes more readily into the bile than it does into the urine. In this connection attention was also called to the work of Hallauer and Gürber[3] who, after the intravenous injection of casein solution into rabbits, recovered considerable quantities of it from the bile as well as from the urine.

While not disputing the possibility of albuminocholia under the conditions of the experiments of Brauer and Pilzecker, it seemed to the author they have not proved that the albumin found in the bile was eliminated by the liver. It is just as possible that it was due to inflammation of the gall bladder and biliary passages which they observed on autopsy. To reduce the possibility of error from this source the writer carried out a number of experiments on dogs, each of which was under ether anesthesia, with a temporary biliary fistula. The neck of the gall bladder in each case was ligated previous to the introduction of a cannula into the common bile duct. The bile was collected and tested for albumin according to Brauer's[4] method. Either amyl alcohol or ethyl alcohol alone or a mixture of the two was injected into the stomach or small intestine. Adequate control experiments were also conducted. The collected bile was tested for albumin in both sets of experiments.

[1] Brauer: *Loc cit.*

[2] Pilzecker: *Zeitschrift für physiologische Chemie*, 1904, xli, p. 157.

[3] Hallauer and Gürber: *Zeitschrift für Biologie*, 1904, xlv, p. 372.

[4] Brauer: *Loc. cit.*

The results obtained were not uniform. Distinct cloudiness on boiling appeared in the bile of one experiment both before and after poisoning with amyl alcohol. On the other hand, in one of the experiments, the bile remained perfectly clear on boiling before and even after injection of amyl alcohol. After the administration of small quantities of amyl alcohol, *e. g.*, 5 c.c., there was no albuminocholia. Following the injection of 20–30 c.c., however, the bile became distinctly cloudy on boiling after slight acidification with acetic acid. In none of the experiments carried out as indicated were more than traces of albumin found in the bile. One special experiment, however, on a dog poisoned with ricin gave a different result. The dog received in three days two subcutaneous injections of ricin, 1 mg. per kilo each time. He was found dead the day after the last injection. The bile removed from the gall bladder showed the presence of a considerable quantity of albumin.

It seems probable, therefore, that the albuminocholia after poisoning with ethyl or amyl alcohol, as observed in animals with permanent fistulas, was due to irritation of the bladder and only slightly to lesions in the liver. The question whether albumin passes more readily into the bile than it does into the urine was also studied. The result in every case showed considerable quantities of *albumin* in the urine after poisoning with amyl alcohol.

A few experiments on rabbits have also been undertaken. Cantharidin or arsenic was injected subcutaneously until albuminuria or hematuria was induced. A biliary fistula was then made and bile collected. In none of these experiments was albumin found in the bile.

RECAPITULATION OF THE NAMES OF THE AUTHORS AND OF THE TITLES OF THE COMMUNICATIONS.

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101. [With **S. J. Meltzer.**] On the effect of magnesium salts upon the excitability and conductivity of nerves.

119. Experiments with some saline purgatives given subcutaneously.

139. [With **S. J. Meltzer.**] A vago-esophageal reflex.

Bardeen, C. R.

138. The effects on embryonic development of the Röntgen rays acting on the spermatozoa of the toad previous to fertilization. [Presented by **Eugene L. Opie.**]

Beatty, W. A. [with **P. A. Levene.**]

104. On the decomposition of purin bodies by animal tissues.

127. On the digestion of gelatin.

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94. Mutation in the evening primrose, *Onagra biennis* (*L.*) *Scop.*

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103. [For **Hideyo Noguchi.**] The action of eosin and erythrosin upon snake venom.

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111. [For **F. G. Benedict.**] The cutaneous excretion of nitrogenous material.

133. [For **Frank P. Underhill** and **Oliver E. Closson.**] The influence of subcutaneous injections of dextrose upon nitrogenous metabolism.

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Granger, A. S. [with **L. B. Stookey.**]

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Hatcher, R. A. [for **L. B. Stookey** and **A. S. Granger.**]

137. The formation of urea.

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Lusk, Graham [with **A. R. Mandel.**]

113. On the intermediary metabolism of lactic acid.

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117. [With **R. D. McClure.**] On the blood-pressure relations in mitral insufficiency and stenosis.

McClure, R. D. [with **W. G. MacCallum.**]

117. On the blood-pressure relations in mitral insufficiency and stenosis.

Mandel, A. R. [with **Graham Lusk.**]

113. On the intermediary metabolism of lactic acid.

Mandel, John A. [with **P. A. Levene.**]

105. On the biological relationship of nucleoprotein, amyloid, and mucoid.

Meltzer, S. J.

101. [With **John Auer.**] On the effect of magnesium salts upon the excitability and conductivity of nerves.

120. [For **Joseph Erlanger.**] The effects of extra stimuli upon the heart in the several stages of block, together with a theory of heart-block.

121. On the nature of the reflexes controlling the successive movements in the mechanism of deglutition.

139. [With **John Auer.**] A vago-esophageal reflex.

Murlin, J. R. [for **Percy M. Dawson.**]

132. The dicrotic elevation at different points of the arterial tree.

Noguchi, Hideyo.

102. [With **Simon Flexner.**] The action of eosin upon tetanus-toxin and tetanus.

103. The action of eosin and erythrosin upon snake venom. [Communicated by **Simon Flexner.**]

134. [With **Simon Flexner.**] Diffusion into colloids and a biological method for testing the rate of diffusion.

Norris, Chas.

96. A case of spirochetal infection in man.

Opie, Eugene L.

122. The enzymes of inflammatory exudates. A study of the enzymes concerned in inflammation and their relation to various types of phagocytic cells.

123. [For **Richard M. Pearce.**] Experimental myocarditis. A study of the histological changes following intravenous injections of adrenalin.

138. [For **C. R. Bardeen.**] The effects on embryonic development of the Röntgen rays acting on the spermatozoa of the toad previous to fertilization.

Parker, G. H.

128. The reactions of amphioxus to light.

Pearce, Richard M.

123. Experimental myocarditis. A study of the histological changes following intravenous injections of adrenalin. [Presented by **Eugene L. Opie.**]

Pratt, J. H.

129. The relation of blood platelets to thrombus formation.

Richards, Alfred N.

95. [For **Reid Hunt.**] On the influence of thyroid feeding and of various foods and of small amounts of food upon poisoning by acetonitril.

136. [With **John Howland.**] The toxicity of indol.

Salant, William

108. Further observations on the effects of alcohol on the secretion of bile.

125. The effect of alcohol on hepatic glycogenesis.

143. Some observations on the presence of albumin in bile.

Stookey, L. B. [with **A. S. Granger.**]

137. The formation of urea. [Presented by **R. A. Hatcher.**]

Torrey, J. C. [with **B. H. Buxton.**]

124. Stable and detachable agglutinogen of typhoid bacilli.

131. Absorption of typhoid bacilli from the peritoneal cavity.

Underhill, Frank P. [with **Oliver E. Closson.**]

133. The influence of subcutaneous injections of dextrose upon nitrogenous metabolism. [Presented by **William J. Gies.**]

Welker, William H.

142. A simple electrical annunciator for use in metabolism experiments, and in connection with filtration, distillation and similar operations. [Communicated by **William J. Gies.**]

Williams, Wm. R. [with **C. A. Herter.**]

98. Experimental hepatic cirrhosis in dogs from repeated inhalations of chloroform.

Wilson, Edmund B.

97. The chromosomes in relation to the determination of sex in insects.

Woodworth, R. S.

99. Color sense in different races of mankind.

EXECUTIVE PROCEEDINGS.

QUOTATIONS FROM THE MINUTES.

Thirteenth meeting.

Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons. October 18, 1905. President Wilson in the chair.

Members present: Adler, Auer, Brooks, Burton-Opitz, Calkins, Dunham, Emerson, Ewing, Field, Gies, Hiss, Jackson,[1] Lee, Levene, Levin, Lusk, Meltzer, Meyer, Murlin, Noguchi, Norris, Park, Richards, Salant, Sherman, Sweet, Torrey, Wadsworth, Wilson, Wolf, Woodworth, Yatsu.

Members elected: Carl L. Alsberg, S. P. Beebe, R. H. Chittenden, P. M. Dawson, W. J. Elser, G. M. Meyer, P. A. Shaffer, Douglas Symmers, L. L. Woodruff.

Fourteenth meeting.

Rockefeller Institute for Medical Research. December 20, 1905. President Wilson in the chair.

Members present: Adler, Atkinson, Auer, Beebe, Brooks, Burton-Opitz, Calkins, Crampton, Davenport,[1] Dunham, Emerson, Ewing, Field, Flexner, Gibson, Gies, Hatcher, Jackson,[1] Levene, Levin, Lusk, Mandel (A. R.), Meltzer, Morgan, Noguchi, Oertel, Opie, Pearce,[1] Salant, Shaffer, Wadsworth, Wallace, Wilson, Wolf, Wood.

Members elected: W. E. Castle, H. H. Donaldson, David L. Edsall, Thomas Flournoy, R. B. Gibson, Walter Jones, A. S. Loevenhart, John A. Mandel, Fritz Schwyzer, Frank P. Underhill, Francis C. Wood.

[1] Non-resident.

Fifteenth meeting.

[Third annual business meeting.]

Physiological Laboratory of the New York University and Bellevue Hospital Medical College. February 21, 1906. President Wilson in the chair.

Members present: Auer, Beebe, Brooks, Calkins, Emerson, Field, Gies, Loeb (L.),[1] Lusk, Mandel (A. R.), Mandel (J. A.), Meltzer, MacCallum (W. G.),[1] Murlin, Opie, Park, Richards, Salant, Shaffer, Sherman, Torrey, Wallace, Wilson, Wolf.

Members elected: Walter R. Brinckerhoff, Warren P. Lombard, B. T. Terry, E. E. Tyzzer.

Officers elected: President, Flexner: Vice-President, Dunham; Librarian, Lusk; Treasurer, Calkins; Secretary, Gies.

Recommendations of the Council. — The following special report of the Council was considered and its recommendations unanimously adopted:

"**Special report of the Council.** The result of the informal vote on the propositions recently submitted by the Council to the members leads us to offer the following recommendations:

"1. Hold five regular meetings during each academic year as at present, with such additional **special** meetings as, in the opinion of the Council, may be necessary.

"2. Restrict the time allotted for the actual reading of papers, exclusive of discussion, to a limit of one and one-half hour. Empower the Council to adjust the number of papers to this limit as closely as possible; authors to state beforehand the length of time desired by them, but ten minutes to be, as a rule, the maximum allowance.

"3. Authorize the Council to give **precedence,** on each program, to communications which have not been presented before any other body and which have to do with investigations essentially experimental in character.

"4. Require communications from absent members to be presented in brief abstract by a member selected by the author or designated by the Council.

[1] Non-resident.

"5. Instruct the Council to report not only on the eligibility of each candidate for membership but also on the desirability of each candidate's election."

Treasurer's report. — The main items of the treasurer's report were the following:

Expenditures, including the deficit of 1905 ($33.16)	$339.96
Receipts	302.59
Deficit	$ 37.37
Increase of deficit	$ 4.21

Sixteenth meeting.

Rockefeller Institute for Medical Research. April 18, 1906. President Flexner in the chair.

Members present: Atkinson, Auer, Beebe, Buxton, Calkins, Dunham, Emerson, Field, Flexner, Foster, Gibson, Gies, Herter, Lee, Levene, Lusk, Meltzer, Meyer, Murlin, Noguchi, Opie, Parker,[1] Pratt,[1] Salant, Schwyzer, Sherman, Terry, Wolf, Wood.

Members elected: Charles R. Bardeen, G. H. A. Clowes, N. B. Foster, J. H. Kastle, Ralph S. Lillie, D. T. MacDougal, J. J. R. Macleod, Robert M. Yerkes.

Amendments of the Constitution. — The following amendments of the Constitution, which had been duly presented at the fifteenth meeting, were adopted by a unanimous vote of the members present:

Amendment X. — Change Article VII (on quorum) to read as follows: "Twenty members (instead of 'a majority of the resident members') shall constitute a quorum for the transaction of business."

Amendment XI. — Offered at the request of the Librarian and intended to abolish the office of Librarian:

(*a*) Eliminate subsection D of Section 3 of Article III. ("It shall be the duty of each member to present to the Librarian one copy of every publication of his researches.")

(*b*) Remove "a Librarian" from Section 1 of Article V. (List of officers.)

(*c*) Strike out subsection C of Section 5 of Article V, on the duties of the Librarian.

Seventeenth meeting.

Laboratory of the Department of Health, of New York (East 16th St.) May 23, 1906. President Flexner in the chair.

Members present: Atkinson, Auer, Dunham, Ewing, Field, Flexner, Gies, Hatcher, Lee, Levene, Mandel (J. A.), Meltzer, Meyer, Norris, Opie, Park, Richards, Salant, Terry, Wadsworth, Wallace.

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Edwin O. Jordan

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